Southwestern Medicine

OFFICIAL ORGAN OF
ARIZONA STATE MEDICAL ASSOCIATION
NEW MEXICO MEDICAL SOCIETY
EL PASO COUNTY (TEXAS) MEDICAL SOCIETY
THE MEDICAL AND SURGICAL ASSOCIATION
OF THE SOUTHWEST

Volume XIII. APRIL, 1929 No. 4

ANNUAL SUBSCRIPTION $2 SINGLE COPIES 25 CENTS

Entered at the Postoffice at Phoenix, Arizona, as second class matter.
"Acceptance for mailing at special rate of postage provided for in section 1103, Act of October 3, 1917, authorized March 1, 1921."

THORACOPLASTY AND ITS ANCILLARY OPERATIONS

FRANK PORTER MILLER, M. D.
Los Angeles, California

(Read before The Medical and Surgical Association of the Southwest, at Albuquerque, New Mexico, November 10, 1928.)

Favorable results in the surgery of pulmonary tuberculosis depend upon great discretion in the selection of cases, early and skilfully executed complete operations and strict attention to post-operative care, including not less than six months' curing, preferably in a sanatorium. There exists among physicians and patients the hazy notion that the operative shock and mortality are terrific, the pain unendurable, and that the survivors are grossly mutilated, and at best are only able to eke out a burdensome existence of a few years at the most. This is greatly at variance with the true status of affairs as understood when one acquaints himself with the present day technic and results. In this dissertation on compression therapy we have permitted ourselves considerable leeway inthat it is necessary to discuss artificial pneumothorax in conjunction with thoracoplasty. Because of the close interrelationship existing in these kindred subjects, one can hardly confine his discussion to either of them alone.

The ideal form of compression is pneumothorax and not thoracoplasty, but, since the latter is the newer mode of procedure, we will limit our remarks to the so-called paravertebral extrapleural thoracoplasty. It is, as its name implies, an operative procedure, extrapleural in character paravertebral because of its location and it involves the resection of a portion of each rib from the first to the eleventh inclusive. The amount of rib resected must be entirely individualized and is dependent upon the amount of compression desired. In attempting to compress a large cavity, the resection would naturally be considerably greater than when a small one is compressed. At times it is necessary in this procedure to do a parasternal operation, but this is not the rule. The amount of rib resected usually approximates from 120 to 140 centimeters.

EVOLUTION

The evolutionary stages prior to the advent of thoracoplasty are particularly interesting, and I will review the most important and epochal steps.

Drainage of cavities has been practiced for the past few centuries with only a small amount of enthusiasm, and only of late years has added impetus been given to this procedure by the work of Sauerbruch. He is of the opinion that, in certain cases where adequate compression cannot be obtained by paravertebral and parasternal means, due to the dense thick walls of the cavity, drainage should be instituted.

In 1880, Carl Spengler resected ribs from under the scapula via a periscapular incision, and named the operation extrapleural thoracoplasty. He operated cases with an effusion, first aspirating and then resecting. Brauer conceived the idea that, to effect a reasonable result, we must have a compression comparable to that obtained with pneumothorax. Instead of minimal resections, as heretofore had been practiced, he removed the entire lengths of the second to the tenth ribs inclusive. His surgeon, Frederich, performed this operation, and it is known as the "Brauer-Frederich operation."

Brauer came to this country in 1908 and reported a series of cases with the prohibitive immediate mortality rate of 27.5 per cent. This report probably dampened the ardor of any enthusiasts that the method may have had in this country. The poor results were due to too large resections, which provoked cardiac failure, and furthermore, his failure to remove the first rib allowed it to act as a fulcrum and prohibit compression.

Lenhartz used the same technic, and, in addition, pressed the apex towards the hilus, and killed nineteen out of twenty patients.

The classical operation of today is that of Sauerbruch, and it is the one most universally accepted, though a few modifications have been added by the different operators. Preliminary to this operation, a radical phrenectomy is done, and after a week has elapsed, the first stage operation is performed, removing a portion of each rib from the eleventh to the sixth. Approximately two weeks following, portions of the remaining ribs are removed, from the sixth to the first inclusive. It is necessary to do the second stage before the ribs have had a chance to regenerate from the periosteum. The number of ribs resected at a single sitting is dependent upon the physical condition of the patient and his tolerance to the operative procedure. Often it is necessary to do the operation in three or four stages. Usually the ribs are resected from below upward, as many observers have felt that this precludes aspiration of infected material. At present it is a much mooted question as to which of the ribs should be removed first. Brauer modified his original operation in 1911 and his subscapular paravertebral thoracoplasty differs in no way from the typical Sauerbruch operation except that he resects greater lengths of ribs from beneath the scapula and allows it to fall in on the lung, exerting maximum pressure over the seat of greatest disease. He does not remove the eleventh rib, nor the first rib, if the apex has retracted below it.

RADICAL PHRENICOTOMY

Phrenicotomy, or simple division of the phrenic nerve in the cervical region, was first suggested by Steurtz in 1911, but little interest was manifested until Goetz and Felix, in 1922, began reporting their observations. They showed that the hemi-diaphragm was not completly paralyzed in twenty to thirty per cent of the cases in which simple section of the nerve was done. This was due to the failure to intercept the accessory branch of the phrenic, which comes off about seven centimeters below the first rib. The most popular method in vogue at present is the "phrenicus exeresis" operation of Felix. It consists of an avulsion of the cervical and upper thoracic portion of the nerve below the junction of the accessory branch, by slowly twisting out the nerve around forceps. Alexander is of the opinion that it is preferable to remove at least twelve centimeters of the nerve.

The result of the operation is an atonic paralysis of the hemidiaphragm, with an elevation of the diaphragm into the chest. The distance which it will rise is dependent naturally, upon the number of pleural adhesions and the amount of pulmonary fibrosis at the base. In the report of Felix he states that the average distance in height between the two sides of diaphragm after radical exeresis is seven centimeters at the end of inspiration and three centimeters at the end of expiration. The diaphragm is normally two centimeters higher on the right side. Phrenicotomy is a relatively simple minor operation. An oblique incision is made along the upper border of the sternocleidomastoideus muscle and the nerve evulsed. Phrenicotomy is valuable in many cases of pulmonary tuberculosis and is preferably an unilateral procedure and particularly when the lesion is a basal one. Since very little, if any, additional work is thrown upon the contra-lateral lung, it does not necessitate the opposite lung being free from activity, and in this respect it differs from thoracoplasty and artificial pneumothorax.

The applicability of this procedure is indicated: First, in early cases of tuberculosis, especially those in which response to sanatorium regime is not satisfactory and in early unilateral cases which cannot get an adequate result by the original means. Second, in those cases with a marked exudative reaction where the condition is bilateral, it is often well to perform this on the side of greater involvement. Third, for chronic type of cases which contraindicate pneumothorax or thoracoplasty, because of adhesions and the lesion is largely basal. Fourth, as a supplementary measure to artificial pneumothorax; it has the advantage that pleural effusions are smaller and fewer in number and the refill time considerably lengthened. Fifth, as a preliminary operation to thoracoplasty. Its advantages preliminary to surgical collapse are (a) easier coughing and expectoration, which reduces to some extent the post-operative risk of aspiration; (b) greater adjustment of thoracic viscera for the consequences of thoracoplasty.

EXTRAPLEURAL PNEUMOLYSIS

This is the separation of the lung and both the visceral and parietal pleura from the ribs and chest wall. The space created by this separation may be filled by various tissue grafts, paraffin, or not filled at all. Pneumolysis is used as an independent procedure, or concomitantly with thoracoplasty.

Because it is practical to strip the pleura to a limited extent only one can readily see its usage is largely found in lesions of a circumscribed area. Bull, Shivers and Archi-

bald perform it routinely in thoracoplasty after the removal of the upper ribs. Other operators do the same if they feel that the compression is inadequate with thoracoplasty.

In event it is not done at the time of the thoracoplastic procedure, it should not be advised for an additional six months, allowing ample time for fibrosis to complete its work following collapse. It has some specific disadvantages: First, the danger of tearing into infected pleura, or lung; second, the fills which are usually fat, gauze or paraffin, may become infected and extruded through the wound or break into a pulmonary cavity by pressure necrosis of its wall; third, pressure exerted upon the mediastinum causing cardiac distress.

OPEN INTRAPLEURAL PNEUMOLYSIS

Open intrapleural pneumolysis is performed at times when adhesions prevent compression with pneumothorax. This is done by ordinary thoracotomy operation and adhesions severed by the fingers or knife. If the adhesions are in the upper portions of the chest, it is usually advisable to remove a portion of the rib and then complete the severance.

CLOSED INTRAPLEURAL PNEUMOLYSIS

Jacobaeus has devised an instrument by which adhesions are severed without the need of open thoracotomy. He introduces two cannulae, under local anesthesia, near the adhesions to be severed and in one he introduces the thoracoscope, and in the other the thermocautery, which burns through the adhesions. When the operation is successful it prepares the patient to receive a total artificial pneumothorax. Unverricht has modified the original instrument as devised by Jacobaeus, and the procedure is usually designated as the Jacobaeus-Unverricht operation. It is not devoid of dangers and disadvantage and it should be borne in mind that string-like adhesions, not too thick, are the ones for which it should be utilized.

The main disadvantages lie in hemorrhage at the time of operation, and empyema following in the wake. Nevertheless, it has clinical application in selected cases, even though it is fraught with danger.

The greatest proponents in this country are the Matsons in Portland. They are using it with a fair degree of success.

INDICATIONS

I can best exemplify the indications of the operation by submitting a case. We have here an individual who has been ill for the past five or six years. He has followed all the prescribed training as taught him in the sanatorium, has persisted intelligently in the cure, and he has reached the point where his condition is stationary. In the beginning of his cure he had a massive lesion upon the left side, involvement from the apex to the base—a condition which was largely exudative in character. Due to the fact that he had some involvement in the upper right, it precluded pneumothorax. He now has maintained his regime for about five years and, it goes without saying, he is getting "darned" tired of his job.

Replacing the exudative process in the left lung, we find a large amount of fibrosis interspersed with air-containing tissue, and the condition now has largely confined itself to an excavation in the upper left lung from which he expectorates about seventy cubic centimeters of purulent sputum during a twenty-four hour period. This remains laden with bacilli. The right side has continued to heal out and its condition is one of apparent arrest. This individual realizes that the majority of far advanced cases pass on to exitus in five to ten years, and he is anxious that something additional be tried. It is an unwritten law that in all cases in which thoracoplasty is anticipated, pneumothorax should first have been tried. It was tried to no avail, and after a preliminary radical phrenicotomy, thoracoplasty was done in a two stage operation. It goes without saying that, in any case, you must have a surgical risk free from complications and preferably afebrile. In the case above mentioned the patient was above his normal weight, allowed to be up eight hours per day, and was walking three miles per day. He realized the potentiality of his health and this provoked the operation.

Another factor should be borne in mind. Prior to the advent of this operation we exercised considerably more patience and struggled along with cases and many were arrested, but today, unless a far advanced case makes a comparatively rapid recovery, he is very prone to be headed for surgery. Indications are almost analogous as for pneumothorax, but more care should be exercised in the selection of cases.

CONTRA-INDICATIONS

As I stated above, these candidates should be free from all complications. No cases of enteritis, bone, gland or joint tuberculosis should be considered. Injudicious selection of cases is placing a very worthy operation in ill repute. Personally, I have never had any good results from cases with bilateral cavitation.

Thearle recently told me of a case in which he effected a result, but he cautioned against it. In view of the fact that his series now has accumulated the immense proportion of 160 cases, it is reasonable to follow his advice. A lesion at the base and considerable trouble in the hilus of the contralateral lung, is a contraindication which is almost universally accepted.

While dwelling upon the matter of contra-indications, I wish to add a note relative to fibrosis. Heretofore, in the curing process, we have craved the formation of fibrosis, but I am of the opinion that fibrosis in excess is detrimental. Where the retractive process is great and a chronic toxemia has been present for many years, we are sure to have myocardial changes, and this is less apt to occur where the fibrosis is only moderate. One should be very careful relative to the status of the myocardium, and with an atrophic heart and blood pressure hovering around 100, reticence should be the foreword. The majority of all deaths in tuberculosis are due to failure of the right heart. Cardiac stress can easily be determined by inspection, as the respiration is labored and the accessory muscles of respiration are all brought into play.

MISTAKES

This patient came to the sanatorium in 1919, with his left chest full of fluid and an extensive involvement throughout the left lung; a small amount of trouble also in the upper right; no respiratory distress was experienced, so the fluid was not molested. He remained in the sanatorium for two years and, upon leaving, his condition was one of quiescence. For the next three years he had his ups and downs with an occasional exacerbation. We performed a two stage operation upon two different occasions, and the result at present is quite comparable to some of the early Brauer-Frederich results. Inability to get the desired result from the initial operation, provoked the removal of additional sections of the ribs, and this patient was operated upon four different times. Because the resections were so great and there was a failure of new bone formation which would ordinarily immobilize the compressed side, we got a flaccid chest, with a resultant paradoxical movement, which swings inward upon inspiration and outward upon expiration—the so-called pendulum respiration which allows stale air from the collapsed lung to enter the uncompressed side upon inspiration and vice versa upon expiration. In this case we defeated our own purpose in attempting to produce rest in a diseased lung. In the first place, we removed too large sections of ribs and the pendulum movement keeps the grossly diseased lung in constant action during the respiratory phase.

Another common form of mistake in these cases is operating upon cases in which the contra-lateral lung does not justify such a formidable procedure. We have under treatment at the present time two cases in which thoracoplasty was done and, six weeks following the operative procedure, the contralateral lung was in such a condition that pneumothorax was instituted. I realize that there are occasional cases in which you will get an extension to the better side after operative work, but the question comes to my mind as to whether these paritcular cases were ever operative risks. In both of these cases the ribs were not resected wide enough and there was a failure to produce compression upon the sick side, and this, in addition to pneumothorax upon the better side, makes their condition very precarious at this time. I look very much with disfavor upon bi-lateral compression. It appears to me that in these cases we are only attempting to increase the longevity of an incurable consumptive.

In my series of cases, another mistake which stands out quite vividly in my recollection is the following: After pneumothorax in 1924, an emypema developed, and, to combat this condition, we did the lower phase of thoracoplasty. Temporary improvement followed for about four months and a year later we attempted to remove the upper ribs and effect a complete compression. Of course we were unable to do this because the lower ribs had become so thoroughly immobilized by the bridge which forms between the two cut ends. This is another reason why the second stage of this operation should be completed before the cut ends have had a chance to bridge across.

COMPLICATIONS AND RESULTS

1. The most common postoperative complication is cardiac failure. Every case of ulcerative phthisis of long standing has more or less of a myocarditis. This can be counteracted by a pre- and postoperative digitalization.

2. Aspiration pneumonia. We can minimize the danger of pneumonia by: (a) Preoperative emptying of cavities; (b) operating in an analgesic state without complete abolition of reflexes; (c) no postoperative opiates, which, in pain-abolishing doses, inhibit coughing and produce retention of sputum with increased toxicity.

RESULTS

The best statistics at present in this country are probably those of Thearle and Archi-

bald. Thirty-five per cent apparently arrested, thirty-five per cent improved, seven per cent immediate mortality and twenty-three per cent late deaths. This twenty-three per cent will die from the progression of the disease irrespective of whether you operate or not. The enormity of this problem can best be visualized by calling your attention to the fact that one thousand individuals die from tuberculosis every week in the United States. By a conservative estimate we have here in the States 30,000 cases where surgical interference is indicated. This represents only three per cent of the total tuberculous population in this country.

THE PRACTITIONER AND THE DIAGNOSIS OF GENERAL PARESIS

NATHANIEL H. BRUSH, A. B., M. D.
Santa Barbara, California.

(Read at the Fourteenth Annual Meeting of the Medical and Surgical Association of the Southwest, Albuquerque, New Mexico, November 8, 9, 10, 1928. This paper, with a few minor alterations, appeared in the American Journal of Psychiatry, Vol. III, No. 4, April, 1924.)

The presentation of a topic dealing with certain psychiatric problems as viewed from the standpoint of the internist, would not be out of place before this Association.
eral practitioner.

In the everyday practice of the internist or surgeon, many problems must arise relating to the recognition and diagnosis of many of the fairly common psychoses. Therefore, it seemed to me that a discussion of the chief symptoms and diagnostic points of a fairly common psychosis, such as general paresis, could well be presented here for the consideration of the internist and the general practioner.

Paradoxical as it may sound, general paresis may be one of the easiest of the major psychoses to diagnose, or it may be one of the most difficult. Often the difficulty arises from the inclination to regard this psychosis as a rarity or a mystery. The ease of diagnosis depends upon several factors: (1) The stage of the disease; (2) the type and rom; (3) the familiarity the practitioner may have with this condition.

It is not my purpose to dwell to any extent upon the pathological anatomy of general paresis, for the practitioner will not be be so much concerned with the finer points in the diagnosis as he will be in the making of the actual diagnosis.

Remembering that paresis has its etiological agent in syphilis and syphilis alone, the internist will derive some help in the knowledge that, just as visceral syphilis is a protean disorder and can simulate any known disease, so paresis can simulate any known mental disorder. But this point should not be overlooked: th earliest symptoms of paresis may be entirely somatic and have no reference to the central nervous system. Keeping these two points in mind, the practitioner can approach the problems in diagnosis with a better viewpoint than if he had not considered them.

Proceeding with the various steps in the examination of a suspected paretic, the examiner will find, contrary to his expectations, that the history may be decidedly misleading. For, when one has had the opportunity of examining a number of paretics, even in the earliest stages before an alteration of character has taken place, it will be noted immediately that many paretics deny syphilis both by name and symptom. A most searching inquiry will not reveal any infection that remotely resembles syphilis.

Even if a suspected paretic admits a specific infection, backed up by the evidence of a genital scar, the history will not, in the majority of instances, bring to light the occurrence of any secondary manifestations. This fact has, at various times, aroused conjecture as to the possible existence of one or more strains of the treponema pallida.

General paresis does occur among the negro race, but not with the frequency that it is found among the white race. This and the above observation regarding secondary lesions, have been used as arguments by those trying to prove the existence of strains of the treponema pallida of varying virulence and selective action. In the negro race, the treponema pallida seems to have a special predilection for the great vessels of the heart. Thus we hear far more frequently of aneurism and aortitis in this race than we do of neuro-syphilis.

In many instances, the onset of paresis is so insidious that the patient's complaints—and often there will be a multiplicity of them—mask the vital underlying condition. This multiplicity of complaints, complaints referred to every one of the bodily systems, is quite likely to throw the physician off his guard and make him think that he is dealing merely with a neurotic or neurasthenic individual.

Many of the standard text-books on psychiatry contain excellent word-pictures of the standard paretic, but it is the paretic who is not "standardized" that causes the greatest number of faulty diagnoses. Often this is not entirely the fault of the physician, but in some instances he may be to blame for not remembering that any syphilitic disease is tremendously protean in its nature.

Again, many text-books have outlined the

classical physical findings of paresis as consisting of: Argyll-Robertson pupils; increased deep reflexes; tremor of the hands, lips and tongue; disordered speech and writing; and possibly some ataxia of the extremities. All of these may be found in the classical paretic, but their absence does not indicate that the patient is not a paretic. This is especially true of the Argyll-Robertson pupil.

More than one physician has told me very emphatically, "Mr. Blank cannot be a paretic, for his pupils react to light." It has been quite true that the pupils did react to light but it was not true that the patient was not a paretic. When present, the Argyll-Robertson pupil can be considered as extremely valuable evidence of a neurosyphilitic affection. But its absence does not exclude this condition. Here is where the practitioner is liable to go off the track. The paretic's pupils, even if they do react to light, wil, if examined carefully, often show some interesting changes. Inequality of the pupils (anisocoria) should immediately stimulate suspicion, as also should any irregularity or notching in their outline. A sluggishness in the light reaction warrants investigation. Some writers lay particular stress on the loss of the consensual reflex and consider it a valuable sign indicative of early neuro-syphilis, and perhaps the forerunner of the Argyll-Robertson pupil.

Too many internists overlook the increased deep reflexes and regard them as merely a manifestation of some psychoneurotic state. In this way another valuable sign may be passed by .

The tremor so often shown in a paretic's hands may not attract undue attention. It may be woven into the above-mentioned diagnosis of a psychoneurosis. or it may be made to fit into the hyperthyroid symptom complex.

Not suspecting the presence of a grave disorder the practitioner will not be on the outlook for speech or writing distortion and may omit the special tests that would reveal abnormalities along these lines. It is well, in suspected cases when no speech or writing disorder is at first observed, to examine on these points when the patient is fatigued.

The ataxia, if present, is less likely to be overlooked for it may be observed as the patient enters the office or may perhaps be noted on other occasions. There are certainly few physicians who, noting the presence of an ataxia, will neglect to give this finding special consideration.

There is still another physical sign which I have neglected to mention up to the present time: I refer to the paretic seizure. This seizure may be easily mistaken for and confused with a variety of other conditions giving rise to convulsive attacks, i. e., uremia, syncope, epilepsy, hysteria, apoplexy, etc., and it has even been mistaken for sunstroke. Certainly the history or observation of a seizure or anything suggestive thereof, should require of the physician a rigorous investigation into the etiology, nor should he be satisfied until he has arrived as nearly as possible at the correct diagnosis.

When a medical student, I many times heard various teachers of mine remark, "When in doubt, think of syphilis." The internist might do well to remember this, not only in connection with bodily ailments but also in dealing with what, to him, may appear to be obscure mental or nervous disorders. For the assistance of the practitioner we might say, "When in doubt suspect paresis, and do not be satisfied until you have exclded it."

All psychiatrists are acquainted with the classical and standard physical signs of paresis and the classical mental picture of the grandiose, expansive and euphoric paretic. This picture has been so well presented in practically all text-books that it has come to be recognized as **the** paretic picture. But when this picture is not present, as is quite commonly the case, the untrained observer may encounter difficulties. At such times it is well to remember that there are numerous other mental symptoms that are distinctly abnormal, to recall that the disorder is a dementing one, and that, in place of complacency and euphoria, there may be a depression of a degree leading to suicidal attempts. If these symptoms can be correctly observed and linked up with other findings, the result may be a correct diagnosis that otherwise might be missed.

How often it is true that paresis, like lightning, strikes when and where it is least expected! The story of many of these cases is the rapid descent from affluence to poverty, from a position of respect and esteem to the gutter and degradation. The unfortunate part of such happenings is that a certain percentage of them could have been prevented, if the family doctor had had the foresight to recognize and to heed the writing on the wall.

Countless examples could be cited of men of wealth and influence, as well as of men in less prominent walks of life, who have first attracted the attention of their friends and their families by petty eccentricities and peculiarities. How willingly the family, and even the patient at times, have accepted the physician's statement that the condition may have resulted from over-work or that it was merely a neurasthenic or some other psychoneurotic state.

But instead of responding to the physician's well-intended but futile remedies, the condition becomes more aggravated. Slips from virtue, increasing alcoholism, carelessness about money matters, neglect and untidiness of the personal appearance, may become at last so prominent as to demand recognition and attract attention to the fact that there is something vitally wrong.

Then it may be too late to save a character that has become morally wrecked and to rescue the finances that may have become entangled in the most preposterous wild-cat schemes.

According to many authorities, the correct diagnosis of paresis is not entirely dependent upon the mental symptoms, and very often the practitioner has nothing but the physical examination upon which to form his opinion. Therefore, I concluded that it might be of value to suggest some few salient points to be considered in the physical and mental examination of a suspected paretic. To simplify matters I have grouped these points under various headings. In this way I feel that more emphasis can be put on the important features of paresis for the benefit of the internist and thus place him on his guard, so that, when the suspected or obscure case does present itself, he will be more adequately equipped to deal with it.

1. **History.** Always question carefully as to any past venereal disease, remembering that an intercurrent gonorrhea may have masked a primary lesion, particularly if the latter be very slight. Any information obtained from the relatives regarding a slump in the personality, increased or unusual irritability, and recent laxness in business methods, a decrease in the patient's efficiency, any financial discrepancies or ventures showing a lack of judgment, and a failing memory, is of primary importance. These apparently simple and easily over-looked changes of character often hold the key to the situation.

Pay particular attention to what the patient himself has to say regarding complaints such as headache, dizziness, various aches and pains, disturbances of vision or of locomotion, and question about his libido and potentia. Furthermore, the history of extremely bizarre behavior, explosive outbursts of violent and uncalled for temper, any convulsive attacks; in short, any deviation slight or otherwise from the patient's usual behavior or character, is warning that must not be passed by lightly.

2. **Physical Examination.** Here the points to be stressed are: Pupillary anomalies of any kind, the presence or absence of tremors of the hands, tongue or lips, the reflex picture and the equality of the reflexes, the special tests for speech distortion and note, in a sample of writing, the presence of tremor, mispelled words and omission of words or letters. The patient will occasionally volunteer, or admit, that he has difficulty in writing and in performing work requiring ready coordination of finger movements. Although placed last in this enumeration of important points in the physical examination, the serology of the blood and spinal fluid is of paramount importance. Often it is the court of last resort. The physical examination of a suspected paretic, must, in the absence of a complete serological examination, be considered incomplete and inadequate.

3. **Mental Examination.** One must not expect the internist to be skilled in the performance of a complete mental status. But there are certainly a few items in a mental status with which the internist should be sufficiently familiar so that he can avail himself of them.

The memory can be quickly and often adequately tested for discrepancies by asking the patient the principal dates in his life, such as his birth, the date of his marriage and age at the time, the ages and dates of birth of his children, the age of his wife, the dates of well known historical events, the names of the last three Presidents, etc. Naturally the patient's answers can be checked up with information received from the relatives.

Sometimes, with very little urging, or under the stimulus of tactful and well-directed questions, the patient will elaborate his delusional system in regard to egotistical ideas or any ideas or delusions of persecution. Once started on this tack, the patient will often produce statements so at variance with known information regarding his personality, his business affairs and his mode of life, that the internist cannot but regard them as distinctly pathological.

To Sum Up. Any of the above physical findings to which I have called attention—the mental status showing memory defects, poor judgment, a moral slump and delapidation of the personality, combined with vague neurasthenic symptoms—any of the above, in no matter how mild a form they may be found, should at once be to the internist a red lantern and should demand a most searching inquiry into the cause and exact nature of the condition.

PEPTIC ULCERS AND ASSOCIATES

FRED A. SPEIK, S. B., M. D., F. A. C. S.
Los Angeles, Cal.

(Read before the Medical and Surgical Association of the Southwest, at its Fourteenth Annual Meeting, held at Albuquerque, N.M., November 8-10, 1928.)

The great problem in the medical and surgical treatment of peptic ulcer is the frequency of the association of lesions of the viscera of the portal lymphatic system.

There is not only a recurrence of the symptoms complex of ulcer of the duodenum and stomach, but serious conditions may arise in any viscera in the portal lymphatic system. "Studies have shown that these viscera are closely connected through the lymphatics—that infection readily passes from one to the other."[1]

The studies of E. A. Graham and especially Scheichi Kodama[2] show in their conclusions that (1) "The lymphatics of the duodenum and gall-bladder are intimately and directly connected." (2) "Some of the lymphatic vessels of the liver, gall-bladder, duodenum, pancreas, and appendix enter the lymph glands which are located about the portal vein." "And that by this explanation it is easy to understand why there exists the frequent association of lesions of the portal system if one thinks of these portal lesions as producing a hepatitis which spreads by the lymphatics."

E. S. Judd[3] emphasizes the fact that cholecystitis is almost invariably associated with a certain grade of hepatitis or pancreatitis or both. These studies emphasize the great importance of the recognition and the further study of the pathological processes in the viscera of the portal lymphatic system.

Clinically, in a review of 156 cases of peptic ulcer treated by the Sippy method at the Pasadena Hospital, we were impressed with the frequency of the association of lesions of the appendix, the gall-bladder, the liver and the pancreas, with duodenal and gastric ulcer.

In a review of the 156 cases treated in the last twenty-one months, the records show:

Appendix removed prior to diagnosis of ulcer	35
Chronic appendix diagnosed during management	28
Appendix out during management	8
Appendix out following treatment	6
Gall-bladder removed prior to diagnosis of ulcer	6
Gall-bladder diagnosed chronic during management	26
Gall-bladder out during management	2
Gall bladder with stones	3
	114

These figures present a total of 114 cases of portal lymphatic lesions in association with 156 cases of ulcer, aside from focal infections elsewhere.

Of this number are cited three case reports, which, since they are complete cases, focus the attention on the lesions of viscera in association. The necropsy reports are attached in two cases, clearly demonstrating associated lesions of peptic ulcer.

Case 1.

Ulcer of lesser curvature with stone and empyema of gall-bladder.

Dr. G. F., age 65, was diagnosed peptic ulcer, Dec. 7, 1927 (See Fig. 1). History of a similar attack eight years prior, associated with jaundice. The size and depth of his ulcer made it seem very probable that it was malignant. There was but slight evidence of shading of the barium shadow around the ulcer and no real filling defect adjacent to it. The general appearance of the patient and the fact that he had free HCl 53 and total 68, gave the opinion that it was benign. He was put to bed and treated by the Sippy method. A second x-ray series, Dec. 19, 1927, showed "Marked progress in healing of the ulcer" (See Fig. 2.) Patient symptom free after the first week.

Fig. 1

A third x-ray series found "Ulcer shadow much smaller." (See Fig. 3.) He left the hospital.

January 23, 1928, at midnight, he was seized with sudden severe pain in the epigastrium, with nausea and elevation of temperature. With the knowledge that his ulcer was healing and the association of portal lymphatic infection, a diagnosis of an acute exacerbation of a chronic cholecystitis was made.

Dr. John Breyer, the surgeon, found the gall-bladder buried in adhesions, swollen, tense and reddish grey; thickening of lesser curvature (area of the ulcer) and the appendix long and fibrous. The laboratory diagnosis found cholecystitis (empyema) with a single stone.

The patient left the hospital February 1, 1928, having gained thirty pounls and "feeling fine."

October 1, 1928, he was seized with severe pain in left shoulder and abdomen. Lungs were negative. Jaundice and tenderness over liver suggested hepatitis. Leucocytes 16,500 with 94 per cent polynuclears. The second day he developed a chill, temperature of 106. A pleural friction rub and crepitation. Rales in the left chest. Pneumonia was diagnosed.

Death the fourth day.

Autopsy, by Dr. Frank Sturdevant, was:

Left pleural cavity: Few small fibrinous adhesions on lower lobe near the base.

Left lung: Healed tuberculosis foci at apex. Small interlobular adhesions. Consolidation of one-third of the lower lobe (red hepatization).

Fig. 2

Right pleural cavity: Old and dense adhesions to lung and pleura throught, congestion and edema of right lower lobe, but no consolidation.

Aorta: Arterio-sclerotic plaques present (narrowed and sclerotic).

Abdominal cavity: Omentum is adherent to the peritoneum anteriorly.

Fig. 3

Liver: Small, mottled, and cuts with increased resistance. Thrombosis of portel vein. A 2 cm. abscess in the upper part of right lobe. A stone in the common duct, measuring 0.6 cm. in diameter. Pancreas edematous and fibrous and blood vessels sclerosed and thrombosed. Thrombosis of splenic artery.

Appendix: Fibrotic.

Stomach: Ulcer scar at the pyloric ring, obstructing pyloric opening, about one-third. On the lesser curvature, midway between the pylorus and cardia, an old healed ulcer 1.2 cm. in diameter (Stomach dilated).

Bladder: Contracted, enlarged plus 2.

Left kidney: Enlarged one-third. Capsule thickened and adherent, strips leaving a granular surface. (Chronic, with an acute parenchymatous nephritis).

Right kidney: Similar to left, except for a small amount of pus in the pelvis. Marked arterio-sclerosis of the abdominal arteries and iliacs.

Summary:

1. Chronic pleuritis (right).
2. Healed tuberculosis of left apex.
3. Embolic pneumonia of left lower lobe with an associated acute pleuritis.
4. Arterio-sclerosis.
5. Acute, subacute and chronic hepatitis, panatitis and slpenitis.
6. Healed ulcer of stomach.
7. Stone in common duct and liver abscess.
8. Portal pancreatic, and splenic thrombosis.
9. Healed gastric ulcers with pyloeric obstruction.
10. Acute and chronic nephritis.

Cause of Death:

Primary:

1. Portal thrombosis.
2. Liver abscess.
3. Stone in common duct.

Contributory:

1. Embolic pneumonia.
2. Acute parenchymatous nephritis.

This illustrates the association of gastric ulcer with cholecystitis.

The right upper quadrant is the surgical site of the abdomen, due entirely to the evil associations of the important viscera of the portal lymphatic system. To get rid of this evil the possibilities of the association of the appendix, the gall-bladder and the pancreas with ulcer of the stomach should be borne in mind.

Case 2.

Duodenal ulcer and appendicitis. Mr. H. B. S., age 48, had "stomach trouble" for six years. Diagnosed: Ulcer. (Ulcer of the duodenum on the lesser curvature, Sept. 17, 1923.) Ambulatory treatment for three weeks yielded no improvement. Hospitalized, and four weeks later was discharged as "improved." Clinically showed ulcer still present but smaller.

Six months later he had an acute attack of abdominal pain. Forty-eight hours afterward he came to the office. White blood count, 30,000; marked rigidity of right lower rectus. "Acute appendicitis" diagnosed and operated upon that afternoon by Dr. H. S. McGee. A gangrenous appendix was removed. Death on tenth day.

CLINICAL DIAGNOSES: Peritonitis secondary to ruptured appendix and operation. Duodenal ulcer.

AUTOPSY by Dr. A. M. Moody: Partial obstruction of ileum with dilatation of small bowel and stomach. Healed ulcer of duodenum. Wound discharging with gutta percha drain. Localized small abscesses in region of cecum.

Case 3.

Duodenal ulcer and pancreatitis. Mrs. S. C., age 65, was diagnosed: Duodenal ulcer with low grade obstruction. Treated at the hospital six weeks. X-ray report, "Marked deformity of the duodenal

cap throughout the series." Repeated urine examinations while in the hospital found "no sugar."

October 1st, 1927, she was free from ulcer pain, and had gained in weight from 109 lbs. to 152 lbs. At this time she was hospitalized on account of obstructive gastic symptoms and sugar in the urine. Blood sugar was high. (300 mg. to 360 mg.) Treatment wth diet and Insulin did not relieve. Gastroenterostomy was done, performed by H. S. McGee. Patient recovered and was discharged March 24, 1928, with normal blood sugar, and has remained so.

Ulcer in association with pancreatitis can cause hyperglycemia. Surgical and medical management of the duodenal ulcer brought about a return to normal.

Although gastro-duodenal ulcers heal under proper medical treatment, we must be constantly on the alert for associated pathology. Intelligent observation, with frequent x-ray examinations, finds that the biggest and deepest ulcers gradually get smaller until they disappear, and the patient is symptom free. However, many cases, in which lesions of the portal lymphatic system exist, may have a return of symptoms or a recurrence of ulcer, because these lesions are foci of infection in the gall-bladder or appendix.

Sippy stated that, in order to treat peptic ulcer intelligently, it is necessary to determine the age, the type, the location and complication of ulcer. It is necessary to go further and determine if there are any lesions of the portal system, such as cholecystitis, appendicitis, pancreatitis, hepatitis or peritoneal adhesions.

The taking out of an acute or chronic appendix does not cure the ulcer—note the number of cases in the table who had an appendectomy before an ulcer was discovered. This is one reason why patients do not always get well following an appendectomy. There is pathology elsewhere.

Patients with foci of infection in the portal lymphatic system, should have them removed at earliest recognition. If physicians are on the alert for associated ulcer pathology, the diagnosis will be made more promptly, and better end results will be had.

CONCLUSIONS

1. The lymphatic drainage of the stomach, liver, pancreas and appendix are anatomically closely connected.

2. In sixty per cent of the cases reported above, gastric duodenal ulcer had associated pathology in the liver, the pancreas, the gall-bladder and the appendix.

3. Healed ulcer will not leave the patient symptom free unless the associated pathology is eradicated.

REFERENCES.

(1) Judd, E.S., Journal Lancet, Oct. 1, 1921.
(2) Surg. Gyn. Obst. August, 1926.
(3) Journal American Medical Assn. July 16, 1921.

DISCUSSION

DR. V. C. HUNT, Rochester, Minn. (opening): I want to compliment Dr. Speik on bringing this subject before the Society. I believe it is one which has not received the recognition which is due it, and the emphasis that Dr. Speik has given this subject should certainly call to our minds the fact that a patient can have more than one condition at the same time.

In the early years of surgery, the appendix was the organ chiefly attacked and if the operator could remove the appendix with an incision of not more than an inch, he was something of a surgeon. He simply removed the appendix and was not inquisitive as to what else might be associated. Now, there is such a definite thing as inter-relationship of infections in the gall-bladder, stomach and appendix. That should always be borne in mind, and it is often very necessary to do a general exploration when a patient is operated upon, so this has given rise to a larger incision. Such explorations have oftentimes found lesions in the stomach and gall-bladder whose presence had not previously been suspected.

This also brings to mind the constant difference of opinion that exists between internists and surgeons regarding the apparent methods of treatment of ulcers. An ulcer is not strictly a medical condition, and it is only through cooperation of the internist and the surgeon that the best results are to be achieved.

DR. F. P. GENGENBACH, Denver, Colo.: In speaking in connection with this subject, I want to pay a little tribute to the diagnostic methods of the Mayo Clinic. A great many years ago I had the pleasure of being up there and, while I was limiting my work to pediatrics at the time, I noticed the cases which went through the Clinic. After all possible diagnostic methods had been taken and had come to the same conclusion, if that conclusion necessitated operation, in the abdominal conditions, after the abdomen was opened (and they always made a very free incision), the operator always looked for everything else except the condition he expected to find. Andthis rule I have applied in my practice with children. Mothers will come in, saying "I came because the child has a swollen gland on the neck." It would be very easy for me to say: "Yes! he has a sore throat, or a cervical adenitis," but it has been my practice, and especially with children who are brought to me for the first time, to strip the child and look for everything else I caan possibly find, and the last thing I look at is that child's neck and throat. I have found that the observation I made in the Mayo Clinic at that time, of looking for every other possible cause of the child's fever, has helped me tremendously. It is awfully easy to see the things that are evident, and quite easy to treat those things, but in doing so you may overlook another condition, or a number of things.

Dr. Speik is to be congratulated upon following up these cases—that is something few of us do. It will be interesting to see his results upon this series of 150 cases published in five or ten years from now. The day of the snapshot diagnostician is past. We know now we cannot see a patient once and know all about the condition, or conditins, causing the complaint, whether it be hyperacidity, vomiting, etc. The stomach is the alarm-clock of the human system and it goes off at the slightest provo-

cation, whether it be a bad sight, a dirty story perhaps, or a blow on the head. It is rather foolish to treat a patient for gastroenteritis, when really a brain tumor is at the base of his trouble. Ulcer was given out readily as a diagnosis in some of these cases, and then later pathology developed lymphadenitis, perhaps, as the cause for ulcer. The diagnosis of ulcer is one thing and the management of it is another. Surely no internist would think of treating a gastric ulcer of a patient who was suffering from pyorrhea, or infected tonsils, but would attempt to clear those things up. He would not attempt to treat gastric ulcer over a long period of time with a chronically infected appendix in the abdomen, but would call in a surgeon for the removal of the appendix; and this is likewise true with reference eto the gall bladder. We have to see our patients again and again, and intelligently observe and study their condition. I know they expect us to look at their tongues and listen to their complaints but in addition we must try to learn their condition before we start treating.

Question: Should a patient with gastric ulcer, without complications, be operated or treated with medication?

DR. SPEIK (closing): I might say in regard to treating a gastric ulcer without complications, that the treatment is essentially medical, because, without complications, it is not necessary to operate, and this is one of the most satisfactory conditions to treat. In other words, it is the complications of ulcer, which make operative procedures necessary.

In regard to pyorrhea, tonsillitis, and focal infections, most men get rid of these conditions. Rosenau said he found that these patients frequently get an ulcer of the stomach, and so the men have been taught to eradicate the tonsils and teeth. I do not think, however, that they have as yet quite come to eradicating the gall-bladder and appendix.

COMPLICATIONS OF POTT'S FRACTURES

DENNIS W. CRILE, M. D.,
and
ARCH SPELMAN, M. D.
Chicago, Ill.

(Read before the Medical and Surgical Association of the Southwest, at Albuquerque, New Mexico, November, 1928.)

In 1768, in his treatise on fractures, Pott first described the injury to the ankle which has since borne his name. He wrote concerning the fracture: "It gives infinite pain and trouble to the patient and surgeon and very frequently ends in the lameness and disappointment of the former, and the disgrace and concern of the latter! I mean the fracture of the fibula attended with dislocation of the tibia."

Nearly fifty years later, the great French Dupuytren, writing of the same type of injury said: "Under the most favorable circumstances they almost always leave deformity and lameness which renders progression difficult or painful."

Since that time considerable work and much writing have been done in an attempt to improve the standard of results obtained in the treatment of ankle fractures. However, because of complications and the variability of individual injuries, and sometimes because of inattention to cardinal principles of treatment, incomplete evaluation of minute complications, we still see poor results. From a purely practical standpoint, too much has been written in an attempt to classify ankle fractures, obviously a difficult task. Some ankle fractures have been classified from an anatomical viewpoint, others from that of the direct etiology or mechanism of injury, and others according to the names of observers by whom they were first described. Considerable attention has been given to the definition of the type of ankle fracture Pott referred to in his treatise, pointing out minor errors in his anatomical and functional description, and some confusion still exists over the precise meaning of the term "Pott's fracture." So far there is no satisfactory classification of ankle fractures and for lack of better term, and because of its convenience and long use, we choose to apply the term "Pott's fracture" to include all fractures of the lower three or four inches of the fibula and to classify associate injuries as complications.

Bizarro of London, in a recent and splendid analysis of 213 forearm and leg fractures states that classic Pott's and Colle's fractures are comparatively uncommon. In our practice, Pott's fracture is fairly common, although I presume its frequency compared to all fractures has decreased considerably as the automobile and machinery have increased during the past decade.

There are many peculiar injuries of the ankle region seen as complications to lower fibular fractures. Among the more common are fractures of the lower end of the tibia and rupture of various ligaments of the ankle joint region. By far the most common associate fracture of the tibia is that of the tip of the internal malleolus. Less common are the fractures of the posterior lip of the tibial articular surface; and occasionally there is fracture of the entire lower end of the tibia or the entire internal malleolus (the last named forming the so called bimalleolar fracture of some authors). Less frequently do we see fractures of the anterior lip of the tibial articular surface.

Some injury to joint ligaments occurs in nearly all of these fractures, the most common being the tearing of the internal lateral ligament from the internal malleolus without injury to the malleolus. Another ligament injury probably less frequent but extremely important, as will be pointed out later, is the rupture of the tibio-fibular ligament and perhaps the splitting of the interosseous membrane, commonly and somewhat

inaccurately known as Dupuytren's fracture.

Less commonly, as complications of Pott's fracture, one sees fracture of the bones adjacent to the tibia and fibula at the ankle, such as the astragalus or subastragaloid bones. Fracture of the astragalus is usually through the neck or body and that of the os calcis through the sustentaculum tali.

It does not seem necessary, here, to deal in detail with the diagnosis of Pott's fracture, but we do wish to emphasize the possibility of overlooking minor bone injuries in this region and the danger in their neglect, and to stress the fact that all ankle injuries should be considered as fracture until proven otherwise. Many disappointing results have followed the treatment of trivial Pott's fractures in the belief that a sprain was the sole injury. The x-ray is the best single means we have for examining for possible fractures, especially when clinical evidence is absent.

It should be borne in mind, in examining skiagrams of the ankle, that, according to the astonishing studies of Geist, more than fourteen per cent of all normal individuals possess accessory bones in the foot and ankle which may be confused with fractures. Those of practical importance, with the percentage of incidence in which they are found (according to Geist), are: Os trigoneum, seven per cent; os tibiale externum, fourteen per cent; os peroneal, seven to ten per cent; os vesalii, one per cent; and secondary os calces, two to three per cent. External and internal posterior astragaloid tubercles sufficiently enlarged to be confused with exostoses, are found in at least fourteen per cent. The identification of such accessory bones is often extraordinarily difficult, especially in the presence of fractures at the ankle. I have seen operations performed for the removal of such normal bones in the belief that they were loose chips. The differentiation between these bones and old rounded off joint chips is sometimes impossible. The stereoscopic x-ray helps, as does an anteroposterior and lateral view. These accessory bones are never free in the joint space and their positions relative to bone or tendon, do not shift, as is apt to be the case when loose joint chips are present. The rarity of Pott's fracture in young individuals, in which the epiphyses are not united, seldom causes confusion; this is in contrast with the epiphyseal shadows about the elbow, which are often confused with elbow fracture.

The reduction of these fractures is a problem chiefly concerned with the mechanism of a mortise joint and the problems of leverage. The principle of treatment of all Pott's fractures and the various bone and ligament complications, is exactly the same in all instances excepting only the uncommon fracture of both malleoli or the even less common fracture in which a large part of the articular facet of the tibia has been carried away.

It is our custom to reduce such injuries as soon as possible after they are seen and to follow this with the application of a plaster cast, the only exceptions being cases in which there has already occurred much swelling of and effusion into the ankle region or in which there is a compound wound. Occasionally we use splint fixation or merely a pillow splint.

The usual disabling deformity in this condition is a spreading of the ankle joint, destroying the normal snug approximation of the astragalus to both malleoli and resulting in an unstable joint which wobbles laterally when weight is put upon it. It is this unstability which causes disabling pain and insufficiency in the later stages.

In the recent case, reconstruction can be accomplished by forcible inversion of the foot, taking advantage of the malleolar attachment of the external lateral ligament to drag back the distal fragment of the fibula. Consequent upon this accomplishment, the medial articular surface of the astragalus is brought against the internal malleolus. In so doing, the strain against the ruptured internal lateral ligament is relieved. Dependent upon this medial relaxation, the torn surfaces of the tibial malleolus or the torn internal lateral ligament (as the case may be) are opposed with east, and can be fixed in place for healing. If there is rupture of the tibio-fibular ligaments or the interosseous membrane, these are likewise held in place, a most important procedure in reforming the mortise.

Another procedure of considerable importance in the reduction is the dorsal flexion of the foot, which will replace posterior lip fragments if such are present and, at the same time, may lock the astragalus in position. If posterior dislocation of the foot is present, this can be reduced by anterior traction on the foot, best done before dorsal flexion is initiated.

We obtain the practical accomplishment of these procedures by placing the patient face down on the operating table and inducing sufficiently deep anesthesia to allow complete muscle relaxation. (Spinal anesthesia provides remarkable relaxation for surgical procedures involving the lower limb.) During manipulative reduction, the surgeon stands on the opposite side of the table from the injured leg and flexes the leg to a right angle at the knee, thus presenting the sole of the foot toward the ceil-

ing. This relaxes the gastrocnemius muscle and allows maximum dorsal flexion of the foot. Maintaining a hold upon the ankle with one hand, the surgeon then holds the toes and metatarsal region of the foot in the other hand and, with firm lateral pressure against the lower end of the tibia, forcibly inverts the foot and applies sufficient pressure downward to maintain full dorsal flexion of the foot. It may be necessary to supplement this procedure with considerable rocking of the foot back and forth, and definite crepitus or preternatural mobility should be felt. It may be necessary also to make medial pressure against the calcaneous.

One cannot overemphasize the advisability of firmness, or even harshness, during reduction, always, of course, avoiding undue trauma, but remembering that a single purposeful and harsh attempt will accomplish more good and cause less additional injury than several insufficient and inefficient manipulations.

As the astragalus is felt to slide medially, and after it comes into contact with the tibial malleolus, pressure of the thumb directed against the tibial malleolus, when it is fractured, will usually cause it to slide into intimate contact with the tibia at its fractured surface. Finally, the foot is brought into the exaggerated pigeon-toed position.

This places the ankle in a position for fixation and the assistant should give his undivided attention to maintainnig this position during the application of the cast. When the patient lies prone with the knee flexed, it is not even necessary that the assistant move his hold on the foot and leg except that he slide his hand along the leg from time to time.

There are many instances in which closed reduction fails to produce satisfactory anatomical reposition, including many of the bimalleolar fractures, some of the lip fractures, and many of those in which there is generalized comminution of the ankle joint impossible to classify. Some of these cases are best treated by immediate operation and in some of them reduction must be delayed for an indefinite period. In some cases it is not possible, by any means at our command, to be assured that full function will result.

We must now consider complications arising during treatment. Among these are ischemias due to hematomas or tight bandaging and occasionally nerve palsy due to pressure upon the peroneal nerve near the proximal end of the fibula. Another complication to be looked for during treatment, is insufficient reduction with a resultant wide mortise at the ankle, a condition which, if neglected, is almost certain to result in limited and painful function. Plantar flexion, eversion or chips of bone interposed between articular surfaces, anterior or posterior subluxations of the astragalus, may also be seen.

General complications sometimes met with are those of cardiac syncope, old age, unmanageable people, diabetes, endarteritis and local luetic conditions. Pathological Pott's fractures are very rare. I am not convinced that systemic syphilis exerts a deterrent effect upon callus deposit (except in case of local gumma). In Pott's fractures, compound conditions are not uncommon, in which we find the internal malleolus, from which a fragment has been broken, jutting through the skin. These may usually be treated as simple fractures after disinfection and reduction. The grossly compound fracture, however, presents a peculiarly difficult problem and cannot be included within the scope of this paper. (See Ochsner's "System of Surgery," chapter on Compound Fracture, by Dennis W. Crile.)

By following these general principles, the recent case of Pott's fracture hardly ever causes of serious embarrassment. In the past ten years, we have seen a good many cases of old Pott's fracture with disabilities consequent upon the persistence of the spread condition of the ankle joint. Most of these occurred in cases presenting some peculiar complication at the time of their primary reduction, and our most interesting work in connection with Pott's fracture has been the surgical reconstruction of ankle joints for these incapacitated people. The problems of such reconstruction are so varied and individualized that detailed presentation here does not seem advisable, but I cannot refrain from showing you a few of the x-ray pictures of some of these complex cases, which I have treated at Augustana Hospital within the past two years. An acquaintance with a few horrible examples is often of utmost value in a physical sense as well as in our moral lives. (Lantern slides of eighteen grossly complicated cases were shown.)

The management of cases of Pott's fracture after reduction is important but of infinitely less importance than is the reduction of the deformity.

Fixation is maintained in the average case for about two months. During this time the patient is encouraged to bear weight upon the injured limb enclosed in its plaster cast. The patient is instructed to keep the leg elevated above the horizontal position whenever (literally) he is not walking upon it.

This routine supplies an automatic method for providing massage and venous or lymphatic drainage and does much to prevent the atrophy of disuse, which one finds present in so many of the unfortunate cases.

While walking, the sound foot should be kept in advance of the injured foot. This is especially desirable during the early stages of recovery after the cast has been removed and this simple rule will do more to prevent and overcome ankle stiffness than any or all other after treatment, with which I am familiar—because, so long as the injured foot is behind the sound foot, while walking, there is a tendency toward dorsiflexion at the injured ankle joint.

The patient is advised to eat vegetables, liver, milk, and to take cod liver oil with phosphorus or alphaden (a preparation of minerals in the percentage found in normal bone). During this interval of treatment, my patients often show mental distress and anguish concerning the appearance of the encasted foot—complaining that it appears to them that the foot is twisted out of shape and fearing that I have over-corrected the foot into a club-foot—and this I have attempted to do. They are readily comforted when one shows them the amount of inversion and varus of which the normal foot is capable. In fact, few medical men realize the degree of inversion which is possible in the normal foot. I mention this in the hope that so simple a statement may encourage us to practice greater inversion in the treatment of ordinary Pott's fracture.

CORTICAL ABSCESS OF THE KIDNEY

VERNE C. HUNT, M. D.,
Division of Surgery,
The Mayo Clinic, Rochester, Minnesota

(Read before The Medical and Surgical Association of the Southwest, Albuquerque, New Mexico, November 8 to 10, 1928.)

Infection of the kidney is usually one of two types: it is transmitted to the kidneys by way of the urinary passages; or it is hematogenous in origin, and carried by way of the circulation from remote foci of infection. The first type is often encountered in the obstructnig lesions of the urinary tract, particularly in prostatic obstruction and retention of urine, and usually accounts for the various degrees of renal insufficiency incident to such urinary obstruction. This type of infection is one to be anticipated, is readily recognized by clinical and laboratory methods, and is usually controlled through establsihed principles of treatment. The acute embolic infections of the kidney, frequently resulting in cortical abscess, are not easily recognized, and often present many difficulties to diagnosis. In the consideration of cortical abscess of the kidney, I wish to discuss, chiefly, its relationship to perinephritic abscess which is renal in origin.

Cortical abscess is frequently seen as an acute exacerbation of chronic infection of the kidney. The infection associated with renal and ureteral lithiasis is often stimulated to an acute process in the event of sudden complete obstruction at the ureteropelvic juncture or ureter by stone, with the development of many abscesses throughout the cortex, which give rise to chills and fever. The development of multiple cortical abscesses, as the result of hematogenous metastatic infection from remote foci of infection, often produces generalized symptoms of infection few of which are indicative, by localization or objective data, of renal involvement.

Unquestionably, during apparent good health many bacteria are present in the blood emanating from remote chronic foci of infection and they are not productive of any symptoms or manifestations of infection, as a result of the ability of the kidneys to filter and excrete them in the urine. Likewise, such organisms may be of a low grade of virulence. The bacteriemia of the acute infections, however, is not always as readily handled by the kidneys.

Brewer, from his experimental data and clinical experience, concluded that, during the progress of any acute infectious disease, numbers of microorganisms find their way into the blood stream and usually are excreted by the kidneys. If the number of these organisms is comparatively small, if their virulence is low, and if the kidneys are in a healthy state, their transit through the renal apparatus does nat give rise to a demonstrable lesion. On the other hand, if the number of organisms is large and their virulence high, or if one or both kidneys are diseased, lesions are produced which may go on to the development of any of the types of renal infection or suppuration. Brewer concluded, further, that hematogenous infection is responsible for the greater number of cases of renal sepsis, and that, even in septic conditions of the lower urinary passages, the concomitant renal disease may be of hematogenous origin.

The frequency with which perinephritic abscess has been seen to occur following severe acute superficial infection, has called attention to the frequency with which cortical abscess occurs following such infections, the development of the perinephritic abscess resulting as an accidental termination of the cortical abscess, instead of spon-

taneous absorption and healing, which, in all probability, occurs in many instances of metastatic embolic infections of the kidney.

Furuncles, carbuncles, osteomyelitis, acute tonsillitis, and so forth, are superficial and remote types of infection which tend to metastasize and form cortical abscesses of the kidney, usually several weeks or months subsequent to their onset; often such metastasis presents manifestations of generalized infection after the original remote infection has entirely subsided. This is particularly true of the superficial infections of the skin, such as carbuncles and furuncles. The infection of the kidney is often extensive and may result in multiple abscesses developing to the extent of involving the major portion of the cortex and an overwhelming generalized infection requiring early recognition and nephrectomy. In many cases of unilateral atrophic pyelonephritis, the kidney has probably been the seat of early extensive involvement. Such extensvie infections, however, are rather the exception than the rule, when they occur secondary to remote superficial infections of the skin, and often, through the ability of the kidney to absorb the abscess and the infectious material, attention may not be directed to the infection until symptoms and signs of perinephritic abscess occur. Israel, Jordan and others, have called attention to the frequency with which superficial infections precede perinephritic abscess.

Pathologists have described cortical abscess as areas in the cortex of the kidney where bacteria lodge and grow, producing at first a circumscribed area of hyperemia or hemorrhage and necrosis, with subsequent gathering of leukocytes, and finally disintegration of tissue and formation of abscess. Such kidneys present to the naked eye, under the capsule or on section, small whitish-gray spots, or in the medulla, streaks which are red, gray or yellow, depending on the degree of advancement of the lesion. Embolic abscesses are commonly present in both kidneys and, by extensive coalescence, may give rise to large abscesses. A case of such metastatic origin was observed at The Mayo Clinic in October of this year.

Case 1.

The patient, a man aged thirty-one years, came for consultation because of a pain over the area of the right kidney. When the history was taken, it was learned that the patient had had a boil on the left wrist, four weeks previously, which had healed completely. The pain in the area of the right kidney first manifested itself two weeks after the boil first appeared; it was gradual in onset and its course was progressively worse without radiation. Chills and fever as high as 102° had occurred the week before admission.

The general examination was negative except for extreme tenderness high in the angle between the twelfth rib and the vertebra over the pole of the right kidney. Urinalysis was negative except for a few pus cells. The leukocytes numbered 22,000. With the knowledge of the relationship existing between such superficial infections and infections of the kidney, a diagnosis of abscess of the kidney was made and exploration advised.

Operation did not disclose perinephritic infection, but when the capsule of the kidney was broken, an abscess in the cortex of the lower pole was found to contain about 15 c. c. of thick yellow pus. The remainder of the kidney did not show evidence of disease. Simple drainage of the abscess resulted in subsequent subsidence of chills and fever with immediate recovery.

This case apparently represents one in which coalescence of multiple small cortical abscesses had proceeded to a large abscess which, as yet, was entirely confined to the kidney. Undoubtedly, if early drainage had not been instituted, rupture into the perirenal tissue, with a resultant perinephritic abscess, would have taken place.

Perinephritic abscess is the usual result when resolution of cortical abscesses does not occur, and unless the abscess becomes sufficiently large to become palpable, much difficulty is sometimes encountered in making the diagnosis. These extrarenal or metastatic types of perinephritic abscess often present symptoms so vague and difficult of interpretation that marked renal injury and general sepsis may supervene before its true nature is determined.

In cases of primary organic lesions of the kidney, such as pyonephrosis, lithiasis and tuberculosis, the urinalysis usually discloses microscopic evidence which leads to investigation of the urinary tract. A strikingly uniform observation, however, has been the absence, in most cases, of microscopic elements in the urine if the cortical abscess was extrarenal or metastatic in origin. Not until the cortical abscess communicates with the calices or pelvis of the kidney, do pus or blood cells appear in the urine. If the cortical abscess is small and does not communicate with the pelvis or calices, pyelographic examination may be negative. In the presence of perinephritic abscess there may not be enough in the pelvic outline to be of diagnostic value, and in a few instances a diagnosis of extrarenal tumor will result. The renal functional tests are of materially less assistance in cases of perinephritic abscess of extrarenal origin than in those of primary origin.

The pain is usually dull and aching in the back over the area of the kidney, often not of sufficient magnitude so that a suppurative process is considered. Usually there is no radiation. Much attention has been directed to the point of tenderness in the cos-

tovertebral angle, which usually may be elicited, but sometimes is not elicited at all or only with uncertainty. In the presence of an abscess around the upper pole in a high-lying kidney, it may be absent. With the development of a tumor in either area, the diagnosis is suggested. Pulmonary complications occur sufficiently often in cases of metastatic perinephritic abscess to mask clinical manifestations, to explain symptoms, and to conceal the underlying cause. Chills and fever are often present in particularly virulent infection productive of cortical abscess; however, after perforation through the capsule, chills are usually not present in subsequent perinephritic abscess unless there are still associated unresolved cortical abscesses. A septic type of fever and marked toxemia without localizing signs or positive urinary or urologic data, may lead to much confusion in diagnosis. The leukocytes are practically always between 20,000 and 30,000. The early diagnosis of perinephritic abscess of extrarenal origin embraces the consideration of all causes of high fever, leukocytosis, and general sepsis. However, the recognition of the relationship of peripheral infection to perinephritic abscess may early call attention to the possibility of such a condition, if clinical examination or the history discloses preceding infection. In order to facilitate early diagnosis, it is important to bear in mind the definite relationship between pre-existing or concomitant furuncles, carbuncles, paronychiae and puerperal infections.

Perinephritic abscess may present signs and symptoms so indefinite that the true nature of the lesion may remain unrecognized for a long time, due to the failure to bear in mind this relationship. This is illustrated by the case of a young man observed in May of this year.

Case 2.

The patient came to the clinic because of pain in the left side of the chest. He had had left-sided pleurisy for about six months, had been treated for pulmonary tuberculosis most of the winter, and was finally informed that he did not have a pulmonary lesion. However, afternoon fever of 102° to 103° had persisted for months, without other localizing symptoms of infection.

The patient was 25 pounds underweight, had fever of 100°, localized moderate tenderness over the left renal area, very little leukocytosis, no evidence of pulmonary disease, and negative urinalysis. Careful questioning revealed the fact that, five to six weeks preceding the initial illness, he had had a series of boils from which he had recovered entirely before the onset of the prolonged illness. This information supplied the necessary link in the chain of evidence of fever, localized pain, tenderness, and so forth, to establish the diagnosis of left perinephritic abscess. Exploration resulted in drainage of several ounces of pus from an abscess around the upper pole of the left kidney. This was followed by rapid complete recovery.

This case illustrates the prominence that pulmonary complications may attain and the thoroughness with which they may be misleading in an attempt to recognize the true nature of the underlying or causative factor. Abscess around the upper pole may lead to early diaphragmatic pleurisy, directing all attention to the chest through subsequent pulmonary involvement as localized bronchopneumonia. I have observed late cases in which direct extension through the diaphragm has resulted in empyema.

Some time ago I reported fifty-nine cases of perinephritic abscess observed at the Mayo Clinic, in all of which the abscess was secondary to multiple cortical abscesses of the kidney. In five of these, single or multiple cortical abscesses were found in kidneys so badly diseased as to require nephrectomy at the time of evacuation of the perinephritic abscess. In none of these was there primary disease of the kidney, such as lithiasis and tuberculosis.

The prognosis in perinephritic, metastatic abscess is dependent largely on the interval between the primary remote superficial infection and the recognition of the renal lesion and its treatment. With early recognition, simple drainage of the abscess is followed by recovery. Rarely is nephrectomy necessary. However, if the time interval is long, general sepsis may become so marked that drainage is of little avail. The following case is an example:

Case 3

A man aged thirty-nine had an infection of the ring finger of the right hand which was accompanied by a septic type of fever. Although the finger had been incised and drained many times, the picture of general sepsis continued and later was accompanied by chills. The chills subsided with amputation of the finger but the fever persisted. Nearly three months after the initial infection of the finger, pain developed high in the right side of the abdomen, followed in a few days by pain high in the left side of the abdomen which continued until the patient's admission to the clinic four months after the onset of the initial infectin.

The patient was emaciated and appeared septic; the temperature was 102°. A huge mass bulged posteriorly in the region of each kidney. The urine contained a few erythrocytes and pus cells. The hemoglobin was 30 per cent, the erythrocytes numbered 2,550,000, and the leukocytes 18,700. The renal functional tests were practically normal. Roentgen-ray examination of the chest disclosed an area of consolidation at the right base. A diagnosis of bilateral pernephritic abscess was made and drainage posteriorly was instituted; a liter of pus drained from the left side and 500 c.c. from the right. The patient's general condition was so poor, and general sepsis so marked, that drainage affected the course very little. Dissemination of the pulmonary infection progressed and death occurred seventeen days later. Necropsy disclosed bilateral cortical abscesses, with extension of the perinephritic abscess to the peritoneal cavity, localized peritonitis, and multiple abscesses of the lungs.

Bilateral perinephritic abscesses are exceedingly uncommon; however, a number of cases are to be found in the literature. The organism producing cortical abscess and perinephritic infection is practically always a staphylococcus.

COMMENT

I have presented this subject for the purpose of calling attention to the necessity of wearing in the hat, so to speak, a picture of a condition which, when all signs and symptoms fail to establish the diagnosis, may be solved by developing a sequence of events emanating from an earlier superficial, apparently inconsequential or miner infection. I would emphasize that a definite relationship exists between superficial infections of the skin, such as furuncles, carbuncles, paronychiae, acute osteomyelitis, acute tonsillitis, and so forth, the subsequent development of renal sepsis in the form of cortical abscess of the kidney or perinephritic suppuration should always receive consideration if there is unexplainable septic fever and leukocytosis, with or without localization of pain or tenderness. It is a "ten strike" when one makes the diagnosis not on the indeterminate evidence at hand at the moment, but on the knowledge of the relationship of the present indeterminate evidence to what the patient had, and apparently recovered from, weeks or months previously.

DISCUSSION

DR. K. D. LYNCH, El Paso, Texas (opening): I think it is rather superfluous to discuss Dr. Hunt's paper, but believe it would be rather impolite not to say a word of praise in regard to his wonderful paper, especially the diagnostic part of it.

These conditions, especially in comparison with other infections we have in the kidneys, are rather uncommonly detected and known, but they come much more frequently than we have suspected.

In 1911, some 17 years ago, one of my patients was seized with an attack like renal stone, the pain being most agonizing. X-ray negative. The kidney was explored and a solitary deep-seated abscess about the size of a quarter was found. The difficulty in diagnosing even the cases in which the symptoms are referable to the kidney is that we have no evidence coming in on the pelvic side of the kidney and cystoscopic examination does not give us very much help, so we are often inclined to doubt that the kidney is infected. However, by a careful examination of the kidney and the history, we have oftentimes been able to make a positive diagnosis.

The solitary abscesses are often easier to diagnose than multiple ones. Occasionally we get just a little blurring in the pyelogram if the lesion is in the lower pole of the kidney and sometimes we get a blurring in the outline. In one of Dr. Vance's cases, when the patient was referred for operation for acute appendicitis, Dr. Vance suspected the case was misdiagnosed. We cystoscoped him and, though the evidence would not seem to indicate any lesion in the kidney, yet a few days later we were able to palpate the lesion in the kidney itself. It was one of those cases we treat expectantly and the abscess finally showed up. I have seen a fair number of these cases—some we have had to operate and relieve and some we could get well without operation. I recall one case, that of a woman where we were very crtain of the condition, and when we operated her later for another kidney condition, sure enough we discovered the scars of the abscess. Some of these kidneys go on to true septic infections. While the kidney substance is infiltrated, there does not seem to be any point of free pus in them; at other times, the whole kidney is hard and swollen and you could almost suspect tumor, but you have the septic infection. The only thing to do in those cases is a nephrectomy, and this we have had to do several times, once in the case of a pregnancy. On looking up the literature, I found one man had reported nine cases in which he did nephrectomy with complete recovery in all.

Certainly Dr. Hunt's outline of the method of going about diagnosing these cases has been very logical and remarkable for accuracy and thoroughness.

DR. HUNT (closing): I have nothing to add except that I appreciate Dr. Lynch' discussion very much and want to express also my appreciation to the Association for the privilege of being with you at this meeting.

ADDISON'S DISEASE

With a report of two cases treated by the Muirhead Regime.

R. J. GROOM, B. A., M. D.
Santa Rita, N. M.

THE SUPRARENAL GLAND

Embryology

The adrenals or suprarenal glands were discovered in 1563 by Eustachious, but their importance was first suggested by Addison in 1855. They are two small triangular glands situated just above the kidneys and anatomically are divided into two distinct parts, the medulla and the cortex. The cortex is derived from mesodermic cells of the genital ridge, while the medulla originates from the intramural ectoderm which gives rise to the nerve cells of the sympathetic ganglia and is a part of the general chromaffin system, so called because the cells have a definite affinity for chromium.

Physiology

Removal of both adrenals, whether at the same time or separately, causes death in most animals. It is questionable just how small an amount of adrenal tissue is necessary to maintain life and it probably depends on the amount of accessory adrenal tissue elsewhere in the body. There are varying numbers of accessory adrenals and, on the destruction of part of the suprarenal glands, these hypertrophy and take up the work of the glands.

The adrenals furnish an internal secretion which acts on (1) the circulatory system, (2) the muscular system, (3) pigmentation of the skin, (4) metabolism, (5) the sexual development. The substance known as adrenalin is manufactured only in the medulla of the gland. The internal secretion of the

gland acts as a hormone on other glands of internal secretion, stimulating some and inhibiting others.

ADDISON'S DISEASE

In 1855, Addison, in a monograph on the disease which now bears his name, defined it as an idiopathic anemia, related to disease of the suprarenal bodies, characterized by a bronzing of the skin and mucous membranes, digestive and nervous disturbances, leading to chronic cachexia and terminating fatally.

Etiology

There has been some difference of opinion as to the etiology of Addison's disease. Three theories have been advanced: (1) a congenital defect in the chromaffin system, especially of the sympathetic ganglia; (2) a lesion of the suprarenal cortex; (3) a combination of the two. The latter is the most feasible explanation. The lesions most often producing Addison's disease are: (1) tuberculosis of the adrenals, (2) syphilis of the adrenals, (3) influenza, (4) pneumonia, (5) malignancy of adrenals, (6) sudden frights or shock. It is found more often in males than females, the rate being about sixty to forty and while cases as young as twelve years and as old as sixty have been reported, it is common between twenty and thirty-five.

Symptoms

The onset is insidious as a rule, although in rare cases the symptoms progress rapidly. The chief characatristics are: progressive muscular weakness, nausea and vomiting, increased pigmentation of skin and mucous membranes, hypotension.

(a) **Gastro-intestinal symptoms.** Loss of appetite, with nausea, acid and vomiting are frequent symptoms. Sometimes there is diarrhea accompanied by cramp-like pains of abdomen and legs; more often, especially in the terminal stages, there is obstinate constipation with uncontrollable vomiting, retracted abdomen, simulating peritonitis.

(b) **Circulatory symptoms.** The outstanding circulatory disturbance is the marked hypotension. The systolic pressure may be as low as 50 or 40 mm. of Hg. The blood shows anemia with reduction of red cells and lowered hemoglobin. There is no edema and rarely arteriosclerosis.

(c) **Psychic and nervous symptoms.** In all cases there is a feeling of prostration and utter exhaustion. There may be a partial loss of memory and intellect. Headache and neuralgia are common in acute cases. Stupor, coma, maniacal outbursts and epileptic convulsions may occur.

(d) **Pigmentation.** This is a bronzing of varying intensity appearing first on the exposed parts of the skin and where there is pressure from clothing or irritation. The first involvement is on the face at the border of the lip, the neck, back of hands, areola around the nipples, the lines alba and the genitalia. Areas of pigmentation appear on the buccal membranes and side of the tongue and rarely in the vagina and rectum.

(e) **Metabolism.** Usually there is loss of weight and finally extreme cachexia. The blood shows hypoglycemia and there is a high sugar tolerance.

(f) **Course.** The course varies as a rule from one to three years. One case reported lasting 17 years.

Diagnosis

Without pigmentation, diagnosis is difficult, but rapid fatigability, extreme weakness, low blood pressure, and gastric disturbance are all suspicious symptoms and, combined with bronzing of skin and mucous membranes, should lead to a diagnosis. In tuberculosis of the adrenals, the x-ray will sometimes show calcareous deposits.

Differenital Diagnosis

In considering a diagnosis of Addison's disease, the lesions causing pigmentation must be ruled out. These include: (1) Pernicious anemia, which gives more of yellow color and is more general than the bronzing of Addison's. (2) Exophthalmic goiter, giving a less marked bronzing and accompanied by other signs, such as increased metabolism, exophthalmia, enlarged thyroid. (3) Jaundice is more acute, general and of a different hue than the pigmentation of Addison's disease. (4) Pigmentation from arsenic or silver poisoning is more general in distribution and gives history as well as a different color of bronzing. (5) Pregnancy —the pigment is less marked and the history is of importance. (6) Bronzed diabetes shows low sugar tolerance and other signs of diabetes.

Other diseases simulating some of the symptoms of Addison's are: malignancy, with its cachexia; pigmentation and gastric and nervous disorders; neurasthenia with gastric disturbance; tumors of the ovary with pigmentation. The most difficult to differentiate are: pernicious anemia, malignancy and exophthalmic goiter. One helpful test in diagnosis of diseases giving pigmentation is the fact that in Addison's disease the systolic blood pressure will be raised about ten per cent by the administration of three grains of adrenalin, while it has little effect in other lesions. Prognosis is always grave, death coming in varying lengths of time, but always certain.

Treatment

The underlying cause must not be overlooked and should be treated as indicated. Rest, general hygienic care, diet and stimulation are important. In addition to these, glandular therapy should be added.

THE MUIRHEAD REGIME

History

In the spring of 1920, Dr. A. L. Muirhead, Professor of Pharmacology at Creighton University, Omaha, presented himself at the Mayo Clinic for treatment for Addison's disease in an advanced stage. Together he and Dr. Roundtree worked out the following line of treatment. Epinephrin was given hypodermically and by rectum three times a day and the whole gland by mouth three times a day up to his maximum tolerance. This Dr. Roundtree has named the Muirhead regime and to date has reported twenty-eight cases so treated. The principle of the treatment is the administration of epinephrin by bowel and hypodermically and the whole gland or cortex by mouth to the point of tolerance, which varies widely with the individual case. The untoward results which may occur are general weakness and tremors with palpitation; from rectal rejection, tenesmus; and from oral administration of the whole gland or cortex, nausea and vomiting and intestinal cramps. The other therapeutic measures employed are rest, moderate exercise, diet, out-door life and sunshine.

Case Reports

Case 1. Woman of 42, married, had 3 children, had had active pulmonary tuberculosis at 20 but no activity for 12 years. For the past year she had been growing gradually weaker until, when first seen, she was bedfast and said even feeding herself was more effort than she felt equal to. She had some intermittent diarrhea followed by periods of obstinate constipation.

On examination, the patient was emaciated, there was deep bronzing of back of hands and forearms, tongue and buccal surfaces. The blood pressure was 62/50.

She made rapid improvement on treatment and at present (four years later) is teaching a country school and walking six miles a day. She is still taking adrenalin by bowel and the whole gland by mouth twice daily.

Case 2. A frightened woman of about thirty-eight came into the office one evening. I had noticed her loitering about the hall for some time before she gathered up courage enough to come into the waiting room. She complained of tiring on the least exertion, and had noticed a discoloration of the back of her hands which was slowly growing more marked. She gave a history of being perfectly well until a year before, when, at dinner at a family reunion, her brother had drawn a revolver and killed her father. The shock was so great that she had been a nervous wreck from then on, and had rapidly lost strength. She had marked pigmentation of the back of hands and wrists, areola of breasts, and slightly pigmented areas on tongue and cheeks. Blood pressure was 64/48. On treatment she made rapid improvement for about six weeks, at which time she returned to her home in Nebraska. I have made several attempts to follow up the case but have never had a reply to any of my inquiries.

SUMMARY

1. Addison's disease is more common than the text books would indicate.
2. Both the adrenals and the sympathetic system bear a part in the pathology of Addison's.
3. There are various other etiological factors than tuberculosis.
4. The Muirhead treatment of Addison's is not a cure for the disease but offers the best results of any method of treatment so far developed.

BIBLIOGRAPHY

1. Jour. Am. Med. Assn. January 31, 1925.
2. Jour. Am. Med. Assn. March 5, 1924.
3. Monographic Medicine.
4. Osler: Practice of Medicine.
5. Oxford Medicine.
6. Sajous: Cyclopedia of Medicine.
7. Tice: Practice of Medicine.

EDEMA OF THE FEET

(Discussion of Case 14181, Case Records of the Records of the Massachusetts General Hospital, New. Eng. Jour. of Med., June 21, 1928, p. 956.)

CASE RECORD

An Irish-American farmer sixty-eight years old entered March 9 complaining of swelling of the ankles of four months' duration.

Eight years before admission the patient had an illness similar to the present one, with dyspnea and edema of the legs, lasting for a few weeks.

For a year or two he had urinated three or four times at night and had had a little difficulty in starting the stream. For a year he had not been quite so "spry" in his work as usual. He had, however, continued to do a great deal of work every day until four months before admission. Then he began to have dyspnea on exertion, orthopnea, swelling of the ankles and high colored, often reddish urine. He continued to do some work until three weeks before admission. He thought that exercise as well as a night's rest caused the edema to go down. When it was marked he had pain in his shins and calves, severe at times, especially at night. He had grown more and more dyspneic and in the past month had begun to have attacks of nocturnal dyspnea waking him from a sound sleep, lasting about an hour and forcing him to sit up to breathe. For six weeks he had had cough, chiefly at night, with a quarter of a glassful of whitish sputum daily. For four weeks he had been taking digitalis. Two weeks ago he had nausea and vomiting for two days, due his doctor said to overdosage of digitalis.

His mother had heart disease and rheumatism for the last twelve years of her life. She died of pneumonia.

Clinical examination showed a man sitting up in bed with slightly rapid but not labored breathing. Skin loose; apparent loss of weight. Small deep bilateral supraclavicular glands. Bean sized inguinal glands. Barrel chest. Moist rales at both bases. Heart fibrillating at a slow rate. Forceful apex impulse seen and felt in the sixth space. Left

border of dullness 15 centimeters to the left midsternum, 4.5 centimeters outside the midclavicular line, right border 3.5 centimeters, supracardiac dullness 8 centimeters. Sounds of good quality. A soft systolic murmur all over the precordium, best heard over the tricuspid area. Blood pressure: right 142 to 128/72; left 145 to 125/75. Pulse normal. Artery walls palpable and tortuous. Liver dullness from the fifth rib to 4.5 centimeters below the costal margin; edge palpable. Resistancce to palpation, probably liver, 6 centimeters below the costal margin, slightly tender. Marked pitting edema of the legs from the thighs down. Skin over the right instep and lower leg red, wrinkled and in places broken. Slight edema of the prepuce. A red area over the tip of the sacrum—non-patent fistula. Prostate large, firm, symmetrical, not tender. Pupils normal. Fundi: right disc margin indistinct, left showed a white lateral crescent; vessels tortuous. Reflexes normal.

Amount of urine not recorded, specific gravity 1.022, cloudy at both of two examinations, a large trace of albumin at both, sediment negative. Blood normal. Wassermann not recorded. Non-protein nittrogen 43 milligrams. Icteric index 5.

Temperature 97.1° to 101.9°, rectal. Pulse 61 to 102. Respirations 25 to 14.

March 12 the blood pressure was 190/105. The following day the heart rate was about 85. At the apex was a fairly loud short systolic murmur, transmitted toward the axilla. The first sound here was of fair quality, the second sound normal. In diastole there was a middiastolic third sound. There was no systolic at the base and diastolic here or along the left border of the sternum. On March 13 there were small shallow ulcerated areas on the third and fourth right toes, painful and tender, partly covered with white dead skin. The entire right foot and lower leg were dusky red and slightly colder than the left. The dorsalis pedis pulsation was felt in the left but not in the right foot. On the left a vessel could be felt to pulsate anterior to the external malleolus. A surgical consultant advised no further treatment.

The patient was so noisy that he had to be transferred to a private room. Plans were made for his care at home, though it was questioned whether he was able to stand the journey. It was thought that possibly digitalis might be responsible for his dementia. He became more and more unmanageable. He made response to morphia but only limited use of the drug was possible because of his respirations. In spite of it he overpowered his attendants several times and got out of bed. March 18 in such an excursion he became hyperactive and fell suddenly to the floor, cyanotic and breathing rapidly. Soon after being put back to bed he died.

Discussions by Group I, of the Yavapai County Medical Society and the Medical Officers of Fort Whipple, at their meeting of Feb. 19, 1929:

DR. C. C. BENEDICT, Whipple, Ariz.

I believe that Dr. Cabot would be fairly well pleased with the history in this case, inasmuch as it seems to me there is considerable information in it. I want to emphasize just a few points which we believe have definite bearing on the diagsosis. It will be noted that eight years before this man's admission to hospital he had an attack of similar nature, which lasted a few weeks and from which he apparently recovered. As a farmer he had been accustomed to hard work every day. About a year before admission he had to let down in the amount of work he was doing because of dyspnea on exertion and edema of the legs. Then about four months before admission he had to do still less work every day; in other words, I suppose he had to stay around the house part of the time and take longer rests. In three weeks probably he had to stop work entirely; perhaps he went to bed. That gives a definite decline in the amount of work he was able to do showing symptoms growing progressively worse, which brings him to the date of admission to hospital. He was in the hospital nine days.

On admission, physical examination showed two rather prominent symptoms, dyspnea and edema. Two causes come to mind immediately,—cardiac and renal. We find later that the urinalysis is almost normal; two examinations show specific gravity of 1.022, a large trace of albumin only, probably no casts because it is said the sediment was negative; amount of urine not recorded. That urinalysis showed no evidence which would contribute to a diagnosis of diabetes or nephritis, although a man of this age, 68 years, could have a low grade nephritis without showing a very marked urinary abnormality. N. P. N. 43.

The edema was not confined to the legs but involved other dependent parts. We must remember that because we will refer to it later.

In regard to his dyspnea on exertion, we believe we will show you, by my colleague's discussion, where this as well as his edema originates. We believe these to be of cardiac origin.

The examination of the chest showed it to be barrel shaped, which is not uncommon in a man of that age and with the history he has given. Auscultation of the chest showed moist rales at both bases, which fact will also tend to confirm our diagnosis of the pathology,—a moisture resulting from interference with the pulmonary circulation.

Examination of the eyes showed, on the right, a cloudiness of the margin of the fundus, and, on the left, a slight crescent, light in color, which is probably the result of hemorrhage a long time before. The vessels around the fundi, or of the retina, were tortuous. The other vessels were palpable and tortuous. However, the blood pressure in an early examination was not high, the systolic being 143, later 190-105, a rather high pulse pressure. It is difficult to say that there was an arteriosclerosis when the blood pressure systolic, went to 90 only rather late. With an arteriosclerotic condition he should really have shown a high systolic pressure at the first examination. However, with the appearance of the retinal vessel and the palpable condition of arteries, we are rather inclined to think he had arteriosclerosis.

Liver. The dullness about 6 cm. below the costal border is undoubtedly liver dullness. The negative Wassermann we believe would rule out syphilis, although that is not absolute; but we have other conditions which are much more likely to account for the enlarged liver, such as interference with artreial circulation. We believe enlargement of the liver is due to passive congestion.

We come, then, to the edema of the lower extremities. Here is a rather rare condition: the edema is apparently the same on both sides but there is a difference in the local temperature of the two legs, the right being a little colder, a little more dusky red below the knee. That means one of two things,—thrombosis or embolism, obliterative arteritis or Raynaud's disease. Let us consider first Raynaud's disease, because I believe it is more easily eliminated. In it we find that a gengrene or black discoloration appears at the tip of the toe and gradually progresses upward along the toe; it is not so in this

case, which shows rather a shallow ulcerative area covered apparently with a little dry, whitish skin. It is also said that the skin on the leg was wrinkled and broken in places. Raynaud's disease, as we understand it so far, is a disease of the artery, but there is a difference in the appearance of the leg or the foot as compared to that of thrombosis. A venous thrombosis will result in swelling of the leg. In Rayneud's disease which is interference with the arterial supply, the result is blanching of the extremity and the foot may be almost pallid. Further, the temperaure will drop, as in this case. In a thrombotic condition of the leg, a natural symptom which I failed to mention, and an early symptom, is pain of varying character and severity. In this case there is no mention of pain, but pain depends upon the rapidity of occlusion of the vessel. If there is slow occlusion so that collateral circulation may be provided, there is apt to be an absence of pain or it may be very slight. If occlusion be rapid there is usually a greater amount of swelling and likewise a greater amount of pain. In this case there is no mention of pain. There is mention made that the dorsalis pedis could be felt in the left but not in the right, which is further indication that there is sufficient pressure on the artery because of the exudate in the surrounding tissues to interfere with transmission of pulse to the skin where it can be felt. We believe, therefore, that Raynaud's disease can be excluded from our diagnosis. Another rather pertinent thing is that history states a surgeon was called in consultation, probably in view of the idea that it was Raynaud's disease, and his advice was that nothing further could be done (probably of a surgical nature), so that we believe the artery was about to thrombose.

We believe we haveampleground for this diagnosis because there is evidence of endocarditis, which would provide for the embolus being dislodged and producing the thrombosis.

Coming to the terminal condition, the dementia, we find it was a stage of what might be called "excitation," as a meningitis. Did he develop meningitis in the last few hours to account for this abnormal excitability? The reflexes were normal and we could hardly have a meningitis of that nature developing and have normal reflexes. However, with the interference with the circulation that existed we believe it possible that there could have been sufficient exudate in the brain, probably in the region of the fissure of Rolando, to produce this confusion. The statement is made that this confusion. might be due to digitalis. While it is common or frequent that digitalis will produce nausea and vomiting in overdose, we could find no instance where a dementia or abnormal excitation would follow.

It is believed, therefore, that this patient had a thrombosis from embolism, involving the right leg; arterio sclerosis and possibly, nephritis.

DR. JAMES H. ALLEN, Prescott, Ariz.

Everything in the history of this case points towards a lesion in the cardio-vascular system. There is very little evidence of any gross kidney lesion. The mere fact that he had reddish urine, we do not believe is sufficient to warrant a diagnosis of nephritis. There were no casts. In a nephritis of any severity there would probably be an increase of non-protein nitrogen, which was 43 milligrams, in this case, a normal amount. We believe the urinary condition was probably due to passive congestion. However, in view of the arteriosclerotic changes that are manifestly present, there may be some evidence of a slight intertitial nephritis found at autopsy.

The symptoms date back over a period of eight years. The history states there were enlarged lymphatic glands throughout the body, which might suggest syphilis of the cardio-vascular system. In most cases of this kind, the base of the heart is primarily involved and in the presence of a negative Wassermann we do not believe there is enough evidence to support that diagnosis.

We believe this man began eight years ago with endocarditis and, at his time of life, this is very frequently caused by a focal infection. A man living in a rural community probably would not take very good care of his teeth and mouth. We have no physical examination at that time, eight years ago, so we do not know what kind of endocarditic lesion he had. But the present examination shows that in addition to dyspnea, orthopnea and edema, he had a definitely enlarged heart, which is undoubtedly evidence of dilatation and hypertrophy and is in part due to the undoubted arteriosclerosis present. However, in addition he had a loud systolic murmur which was heard best at apex and transmitted toward the axilla. This we believe is very good evidence that the man had endocarditis involving the mitral valve. which became thickened and shortened. and probably later on there was stenosis along with regurgitation. Apparently he responded to therapy and was able to get compensation. which persisted up to a year before admission to hospital when he began to fail. With an incompetence of the mitral. you have during the contraction of the left ventricle a flow of blood not only through the aorta but back into the left auricle. which puts increased strain on the heart. causes dilatation of the left auricle and subsequent thickening of the wall of that chamber. This. in turn. produces an increase in the size and possibly the number of muscle fibres of the left ventricle. As long as this hypertrophy can compensate for that flow of blood coming back. just so long will that individual be free from symptoms. But let this compensation fail. and there will be a damming back of blood in the pulmonary circulation and resulting pulmonary congestion. This is evidenced in this case by the rales heard at the base of both lungs. This damming back process evidently occurred and the congestion extended back from the lungs and involved the systemic and portal circulations as evidenced by edema and enlargement of the liver Another complication is that of auricular-fibrillation. which is present in this case. In the normal heart the impulse starts from the sino-auricular node and passes to the auricle and thence over the auriculo-ventricular bundle to the ventricles, producing an orderly and regular heart-beat. In auricular fibrillation. the impulses start from multiple foci in the auricles. and many of them are so weak that very few get through into the ventricle. This produces a very rapid and irregular heart action. It seems that this increases the tendency to loss of compensation in the heart. Auricular fibrillation is very frequently found in mitral stenosis and inasmuch as that is very frequently a complication of regurgitation, we believe there is more than a possibility we have it here. Also we know that there is no heart lesion with a greater tendency to throw off emboli. In addition to the fibrillation. which can cause this loss of compensation. we have arteriosclerosis which causes an increase in the back pressure, thus making it harder for the heart to work.

We believe the diagnosis in this case is purely cardiovascular. (1) Arteriosclerosis. (2) Chronic endocarditis with mitral regurgitation and stenosis. (3) Auricular -fibrillation. (4) Dilatation and hypertrophy of the heart.

Discussion at the Massachusetts General Hospital:

RICHARD C. CABOT, M. D.

NOTES ON THE HISTORY

1. "Exercise as well as a night's rest caused the edema to go down." That is a thing that we are struck with sometimes in taking histories of cardiac patients. Ordinarily the rule is the other way; exercise makes the edema come. There are some patients—and I have not been able to distinguish of what type they are—who walk off edema instead of walking it on. That seems to be the case here. Ordinarily when the patient gets up in the morning the edema comes and when he goes to bed it goes off. But sometimes a patient can get rid of edema by using his muscles.

2. "Pain in his shins and calves." I suppose that is from the same cause that makes edematous parts so tender. When you are feeling along the shins you will find that every now and then the patient winces. There is tenderness there more than elsewhere. I suppose it is due to some physical or chemical change in the nerve endings from the presence of the fluid.

3. As the mother's heart disease and rheumatism occurred in the last twelve years of her life we probably can infer that it was not the rheumatism that produces heart disease but the stiffness and soreness about the joints which old people have, and which have no connection with heart disease.

NOTES ON THE PHYSICAL EXAMINATION

1. The right and left blood pressures were identical. Those differences are less than any we can measure wtih any blood pressure machine that I know. I suppose that they were thinking of the possibility of aneurysm. That is the only condition in which we get big differences of blood pressure on the two sides.

2. The red area over the tip of the sacrum is a danger signal for a bedsore. If that is not very carefully taken care of, shielded by "doughnuts" and things of that kind, you do not escape the bedsore. Where else should we look for red areas?

Students: On the ankles—heels—scapulae—great trochanter of the femur.

3. Dr. Cabot: There was an opening into which they could put a probe, but into which they could not get far. I should say it was not a fistulous opening, but a pilonidal sinus.

4. What do you infer from the fundi?

A Student: Arteriosclerotic changes.

Dr. Cabot: With or without hypertension.

5. The laboratory findings are all negative.

6. If we were certain that he had a third sound and not a murmur we could infer nothing. It is one of the most difficult things in ausculation to tell a third heart sound from a mid-diastolic murmur. Very few practicing physicians that I know can do it. It is quite a possible thing to teach, but it did not come up as a thing necessary to teach in our medical schools until recently. We have no positive evidence here either at the apex or anywhere else of a diastolic murmur.

7. If the man could not stand the journey home he could not stand amputation. It was considered and refused.

8. That death again is one of those incidents that you would much rather have happen in an institution than outside. If it happened outside it would certainly be said that the nurse murdered him.

DIFFERENTIAL DIAGNOSIS

From a purely medical point of view I do not think the case offers much difficulty. We have been given the facts on the heart and we have to say there is hypertrophy and dilatation. We know that hypertensive heart disease at his age is overwhelmingly more common than any other type of heart disease. Hypertensive heart disease with cardiac hypertrophy and dilatation are the fundamental things. Failure of the heart in that condition is what gives the dyspnea and the tender condition and edema of the legs. We have to account for that arterial embolism which I have no doubt he had in the right leg. We have got to suppose the possibility of an intracardiac thrombus in the left ventricle. Experience shows that these arterial thrombi usually extend farther up than we expect. We have evidence only below the knee. Unless I am mistaken I think we shall find that his goes away up into the pelvis.

What can we say about the kidneys? I think they will be found to be arteriosclerotic. There is no good evidence of nephritis. There is good gravity of urine, a negative sediment, a fairly low nonprotein nitrogen. I should say the kidneys ought to show general arteriosclerosis but not nephritis.

A Student: Was the sugar test done?

Miss Painter: The urine showed no sugar.

Dr. Cabot: As to his brain we will not argue, as Dr. Mallory did not get permission to examine the head.

The lungs should show general passive congestion and hydrothorax, although we have no evidence of the latter.

The liver of course will show passive congestion. Presumably there will be some fluid in the peritoneum.

A Student: Do you have to suppose an arterial embolus in that leg?

Dr. Cabot: No, you do not. You can have peripheral arterial thrombi with no source found. You always wonder if you have not missed something. I am right in that, am I not, Dr. Mallory?

Dr. Tracy B. Mallory: Yes, sir.

A Student: Is a concentrated urine red, as it says here?

Dr. Cabot: Yes, reddish.

A Student: What was the leading cause of death?

Dr. Cabot: Exertion in a patient with a heart having very little marginal reserve.

A Student: Do you think that it is necessary to assume cerebral thrombosis or meningitis or anything of that sort?

Dr. Cabot: I do not care to spend time on that. If the head had been examined it would have been worth while to go into it I do not believe he had meningitis. I doubt if he had anything more than the arteriosclerotic process; that could account for all the mental symptoms. Patients have to be put into insane asylums for pure cerebral arteriosclerosis.

A Student: How far may renal arteriosclerosis go without being called nephritis?

Dr. Cabot: As far as the pathologist allows. That is up to the individual expert. I am predicting what Dr. Mallory will say. You have to know your pathologist, however.

CLINICAL DIAGNOSIS (From Hospital Record)
Arteriosclerosis, general and cerebral.
Congestive failure.

DR. RICHARD C. CABOT'S DIAGNOSIS
Hypertensive heart disease.

Hypertrophy and dilatation of the heart.
Embolism of the right leg.

ANATOMIC DIAGNOSES

1. **Primary disease.**
   Cardiac infarction.
2. **Secondary or terminal lesions.**
   Bronchopneumonia.
   Arteriosclerosis, generalized (including pulmonary).
   Passive congestion, general.

DR. MALLORY

The most important finding in the case was an immense heart weighing 840 grams. Both cavities took part in the hypertrophy, the left however considerably more than the right. The entire apex of the ventricle was destroyed by a large infarct nearly 5 centimeters in diameter. Over this a thrombus was adherent on the endocardial surface. There was no change in the epicardial surface. A portion of this thrombus had broken loose and produced the embolus in his leg. It is also quite possible, although we have no evidence, that an embolus in the cerebrum may have been the immediate terminal cause of death.

The descending branch of the left coronary artery showed very marked sclerosis, with at one point a small calcified mass which almost completely—not quite—occluded the lumen of the vessel. There is always the possibility in these cases, of course, that a thrombus has occurred at some previous time and has become recanalized, so that in many of these cases of long-standing infarct of the heart we do not find completely occluded coronaries. The infarct here was of long standing, since marked fibrosis had occurred. The heart muscle had been for the most part absorbed, thinning the wall of the ventricle at the apex down to less than two millimeters. At this point it bulged out in the form of a small but quite definite aneurysm of the heart itself.

Thel ungs showed a slight degree of emphysema, and microscopically a few areas of bronchopneumonia were found, but not enough to have been of much significance in the clinical picture.

The rest of the organs showed a marked chronic passive congestion.

The kidneys showed only a moderate degree of arteriosclerosis, many changes in the larger and medium sized vessels, very few in the glomeruli themselves.

I think in answer to what Dr. Cabot was saying as to when we can call vascular changes in the kidney nephritis, there is no hard and fast rule. Every pathologist does have his own criteria. Possibly the chief point is whether there is a more or less generalized process in the glomeruli. With arteriosclerosis of the large vessels only, clusters of glomeruli supplied by the single vessels, which have become thrombosed and occluded, undergo a slow type of infarction, and we get fibrous scars. In general that type of fibrous kidney seems to produce no clinical symptoms other than possibly hypertension. When, however, one has a diffuse process affecting to a slight degree only perhaps, all the glomeruli, we have signs of renal insufficiency at one stage or another. In this kidney there was practically no change in the minute arteries, the afferent arteries of the glomeruli, and very few changes in the glomerular capillaries. It is a case in which I should feel quite confident in saying from the histologic section only that he would not have shown any true renal symptoms other than hypertension.

Dr. Cabot: How far did the embolism extend in the artery?

Dr. Mallory: It was down below the thigh.

Dr. Cabot: This man had a cardiac infarct. What is the ordinary symptom of cardiac infarct?

A Student: Pain.

Dr. Cabot: Yes; and he did not have that. It is not extremely rare to have a case of cardiac infarct without the only symptom by which we can distinguish it. If there is no pain we cannot make the diagnosis. The very large majority of cases that I have known have had pain. If this man had it he did not tell anybody, or nobody told us. Was this a matter of months?

Dr. Mallory: Yes; it was two months at the very least.

COMA—CAUSE?

(Discussion of Case 14341. Case Records of the Massachusetts General Hospital, New Eng. Jour. of Med., Oct. 11, 1928, p. 736.)

CASE RECORD

A married West Indian negress thirty-five years old was brought to the Emergency Ward June 7 in coma.

A month before admission her sixth child was uneventfully delivered. She was unusually slow this time in regaining the loss of weight which was usual during her pregnancies. Five days before admission she developed throbbing fronatl headaches which disappeared in two days after taking aspirin. The day of its onset she had a sense of constriction in the chest. The morning of June 5 she felt dizzy, and on trying to walk to the bathroom fell downstairs, bruising her right arm and cutting her foot. After five minutes she was able to go back to bed. She was alone in the house. That morning a physician found her blood pressure high. All that day and the next she was dizzy. For two days she had not been able to move her right arm. The morning of June 6 her breath had a bad odor. Since that morning she had not urinated. That night she went into coma from which she could not be roused. The physician found her blood pressure still high.

There was no known history of familial disease. The patient had had small-pox. During an epidemic she was in bed two weeks with influenza. Her general health had always been good. All her six children were living and well. She had had no miscarriages. Her husband denied venereal disease in himself and his wife, though he admitted several exposures.

Clinical examination showed a rather obese colorful woman in coma from which she could not be roused. Lungs clear, Apex impulse of the heart not seen or felt. Percussion measurements not recodred Heart otherwise normal. Blood pressure 175/115. Abdomen normal. Vaginal and rectal examinations not done. Right arm maintained in flexion; extension resisted. Left arm normal. Neck not stiff. Right knee-jerk and other reactions to stimuli of the right leg less than those of the left. Bilateral Babinski. Kernig positive on the left.

Amount of urine not recorded. Both of two specimens dark red, specific gravity 1.034, a trace of albumin at both examinations, sediment of one specimen loaded with leuocytes, red blood cells and casts A slowly centrifuged specimen showed 60 to 70 leukocytes per field, 3 to 4 red blood cells and occasional casts. Blood: hemoglobin 90 per cent, reds 5,690,000. Nonprotein nitrogen 43 milligrams.

A lumbar puncture was done the day of admission. The third lumbar space was entered with slight difculty, as the patient struggled, 5 cubic centimeters of bloody fluid was withdrawn which on centrifuging was very faintly, intial pressure about 400, pulse and respiratory oscillations normal, other dy-

namics not done; alcohol and ammonium sulphate positive, 25,750 red cells in the first tube, 24,000 in the second, 22,750 in the third, many crenated white cells; 18 per cent polymorphonuclears, 12 per cent lymphocytes, 2000 large mononuclears, total leukocytes 42 Goldsol 1122332100. Total protein 29. Wassermann negative. At a second puncture the same day 30 cubic centimeters of fluid was withdrawn slowly, bloody as at the first tap. The fluid was withdrawn until the drops came out slowly. Initial pressure 400, final pressure 5 to 10.

Portable x-ray films of the skull showed no fracture, but were not entirely saistfactory.

Nine hours after admission and several hours after the first lumbar puncture the patient had stertorous breathing and was generally spastic. The pulse went up from 60 to 120. For two hours there were alternate periods of this condition and of her previous quiet coma. The pupils were now both dilated and fixed and neither arm nor leg was spastic. Eleven hours after admission the secodn lumbar puncture was done. Ten minutes later the patient sudednly stopped breathing in inspiration and entered a terminal phase of absolute respiraotry paralysis. Five minutes later the heart stopped beating.

Discussions by Group II, of the Yavapai County Medical Society and the Medical Officers of Fort Whipple, at their meeting of Feb. 19, 1929:—

DR. CLARK B. DEVINE, Whipple, Ariz.

The presenting symptom in this case is the coma. Until this woman entered the hospital, only three important symptoms had been obtained in the history—headache, dizziness and increased blood pressure. This emphasizes the fact that a history is of vital importance in the diagnosis of the cause of coma; generally the failure to diagnose the cause of coma properly is due to inadequate history.

The cause of this dizziness and headache is probably the gradual seepage of blood into the brain tissues. That there was blood in the cerebro-spinal fluid for several days is shown by the fact that it was slightly hemolyzed. The history states that the prenatent liquid in the centrifuged specimen was slightly yellow due to hemolysis.

The chest and abdomen are practcially normal. There were a few neurological findings which also pointed to the brain as a point of insult. The right arm was flexed and resisted extension. On the right side the knee jerk and the reaction to stimulus was less than on the left; that might mean there was no response or very little on the right and a little more on the left, or it might have been normal on the right and increased on the left. It is impossible to speculate much on the initial lesion in the brain. With the cerebro-spinal fluid bloody, increased pressure and globulin positive, increased cells and a luetic curve to the gold solution, ordinarily this would mean a suspicion of syphilis, but the negative Wassermann probably rules that out. The increased cells and increased globulin are probably due to septic meningitis caused by the presence of the blood in the cerebro-spinal fluid. Embolus and thrombosis will have to be mentioned in a woman who is recovering from the puerperal condition, but in the case of the plugging of a vessel with a clot of some kind, unless a large vessel is involved, the coma that is produced is very much less than in hemorrhage; also the cerebro-spinal pressure is not much increased and there is very rarely blood in the spinal fluid.

Tumors should be mentioned—tumor causes increased pressure,—but of the triad of headache, vomiting and choked disk, only the first is present in this case and without the other two we would not be justified in thinking strongly of tumor, although one could imagine cystic tumor with hemorrhage within it,—a rare condition.

This woman, after she had been eleven hours in the hospital, became markedly worse, breathing became stertorous and she became generally cyanotic, with pulse (increasing) from 60 to 120, pupils fixed and dilated. Another lumbar puncture was done; about ten minutes later her respiration became paralyzed in inspiration and soon the heart stopped beating. We believe the lumbar puncture had quite a little to do with her death. A pressure con undoubtedly formed when they removed the pressure from below by the lumbar puncture that allowed the pressure above to push the medulla down into the foramen magnum, either by direct pressure or by interference with the blood supply, causing paralysis of respiration. Usually, when a spinal puncture is to be done, where there is tremendous pressure in the brain, a decompression is first done by going into the lateral ventricle and removing enough fluid to lower the pressure to such an extent that a lumbar puncture may be done and the fluid removed in safety.

We believe we have a case of cerebral hemorrhage.

DR. ROBERT S. FLINN, Prescott, Ariz.

Coma appears to be the presenting symptom. Coma is defined as a state of deep unnatural sleep accompanied by irregular or stertorous breathing and often ending in death. It may be caused by a variety of agents and may be roughly divided into two general groups; Group A, in which it comes on late in the disease and there are other symptoms which usually point to the diagnosis; Group B, in which it comes on early in the course of the disease and is often the presenting symptom.

Under Group A we may note the terminal phases of certain infectious diseases, as typhoid, malaria, malignant endocarditis and influenza; the terminal phases of certain infections of the cerebro-spinal system, such as encephalitis lethargica, suppurative meningitis; and also less acute lesions of the central nervous system, such as brain tumor, cerebral abscess and syphilis of the brain. Coma coming on late is also seen in diseases such as uremia, diabetes and Addison's disease.

In Group B, where coma comes on early and is a presenting symptom, we think first of trauma, cerebral concussion, vascular disease of the brain; then of embolism, hemorrhage, thrombosis either artificial or of a venous sinus such as the superior longitudinal or the cavernous; and then certain drugs and poisons such as alcohol, opium, morphia and veronal. Coma also comes on early in heat prostration, excessive cold, excessive loss of blood from ruptured tubal pregnancy, hemoptysis, intestinal bleeding and ruptured aneurism; also in hysterical trance and like disorders.

I think in this case we can eliminate those under Group A and restrict the diagnosis to those under Group B in which the coma comes on early. Let us consider the cause in this group—vascular disease of the brain such as embolism or thrombosis, concussion or fracture of the skull. The other conditions are lead poisoning, alcoholism, heat prostration, excessive loss of blood, etc., which we believe can be easily eliminated. We have decided the lesion in this case is probably due either to fracture of the skull or vascular disease of the brain.

Let us consider the cerebro-spinal fluid of which the normal is described as crystal clear fluid under pressure of from 12 to 15 c.c. of water; contains a very slight amount of globulin which is in-

creased in inflammatory conditions. In practice, ammonium sulphate added to the spinal fluid becomes opalescent if the globulin is increased. Normally the spinal fluid contains a very few lymphocytes, perhaps from 1 to 1½ per cubic m.m.

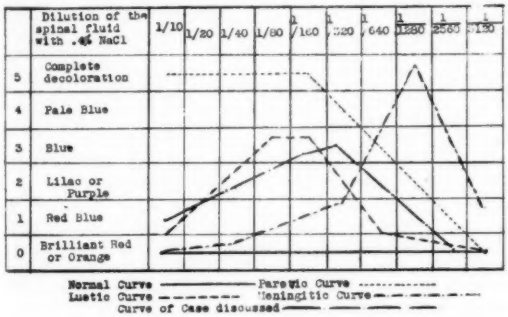
We can regard this fluid as patholigical. You will note that 5 c.c. of bloody fluid was removed and that the fluid was bloody in three specimens. This would point to the fact that the blood was already present in the spinal fluid and not the result of faulty technic. In hemorrhage from the meninges blood appears almost immediately in the spinal fluid and the spinal fluid collected in three tubes is uniformly red.

Analysis of the spinal fluid does not permit us to say whether a meningeal hemorrhage or a ventricular hemorrhage has occurred, because of the extremely rapid diffusion of blood throughout the spinal fluid. Blood may be seen in the spinal fluid following meningeal hemorrhage, particularly in syphilitics, and following an intra-ventricular hemorrhage from the basal nuclei.

It is quite impossible for me to attempt to interpret the two thousand large mononculars in the blood in the face of the total cell count of only 42 leukocytes.

The pressure was tremendously increased, over 400, (normal 12 to 15).

Wassermann was negative and the gold chloride curve was in the paretic zone.



I have indicated on the board in a rough way (see chart) the typical goldsol curve. The gold chloride test of Lange is performed with a solution of gold chloride, potassium carbonate and formaldehyde. The control tube is salmon red in color. When dilutions of spinal fluid are added to the normal tube, we record the change in color according to this numerical marking; No. 0 no change, No. 1 red-blue, No. 2 lilac or purple, No. 3 blue. No. 4 pale blue. No. 5 complete decoloration.

0000000000	a	typical Normal curve
5555542100	a	typical Paretic curve
0123320000	a	typical Luetic curve
0001224531	a	typical Meningitic curve
1122332100		case under discussion.

You will observe that the case under discussion falls in the luetic zone and I suppose we should consider syphilis. However, the Wassermann is negative and the spinal fluid contains blood. An important consideration in the coldsol solution reaction is that minute traces of blood accidentally present will almost invariably give erroneous readings. It appears then that we must disregard the findings in this ase.

The history states that a number of red and white cells were found in the urine and a trace of albumin, specific gravity 1.034. However, the non-protein nitrogen was normal and we are not justified in stating that there was any marked kidney damage on these findings, although I shall not be surprised if the kidneys show arteriosclerotic changes.

DR. H. T. SOUTHWORTH, Prescott, Ariz.

Dr. Devine, in presenting his part of the case, has called attention to the faulty reflexes which this patient had, has given an interpretation of them and has spoken of our inability to localize the lesion within the brain. Thrombosis has been mentioned and eliminated, also tumor. Dr. Flinn has given his interpretation of the spinal fluid, showing why, although this fluid is in the luetic zone, it has very little to do with our case. He has also interpreted the blood chemistry and urine and has given the reason why we think that uremia had nothing to do with the coma. By the same reasoning meningitis of any kind, excepting possibly secondary, is also ruled out. He has told something of the blood in the cerebro-spinal fluid which enables us to say definitely that this patient had a cerebral hemorrhage.

At this time I should like to say that we have found this a very interesting case to study. Had this patient had coma without a fall, it would have been interesting; coma following a fall and without prodromal symptoms, it would have been interesting; but since she had the two together it has been intensely interesting.

Let us discuss as the cause of coma, cerebral hemorrhage as a result of apoplexy. There is much against apoplexy as cause; this woman was only thirty-five years old and it would be rare to find it in one so young, and then it would probably be a result of prematurely atheromatous vessels. We know she had hypertension before this; a physician who saw her prior to admission to hospital said she had a high blood pressure—how high we do not know; after admission to hospital it was 175/115, but that would hardly justify saying she had arteriosclerosis. How many are walking the streets with a pressure up to 250 and having no cerebral hemorrhages? It is possible they could have that hypertension without atheromatous blood vessels. In addition to her hypertension she had a vertigo and one day some findings in the urine which might point toward that condition of the brain.

Syphilis is ruled out, at least we are not justified in mentioning it. Embolism could be thought of, but there is nothing in the heart, nothing in the history and no findings. A brain embolus is usually caused from a vegetative valve in the heart. This brings us to the question of injury to the head. She was sick, complained of a throbbing headache relieved by aspirin. Two days before admission she had a dizzy spell and fell down stairs, bruised her right arm and cut her foot. After five minutes she got back to bed; we don't know whether she was unconscious. The mildest form of injury to the head is that of concussion. If she had concussion following an injury to her head it passed off very rapidly. The next class of injury, in severity, to the head, is laceration or contusion of any grade. It was not an injury of this kind. Let us consider fracture; we have no escape of cerebro-spinal fluid from the ears or nose, no bulging of the pharynx indicating hemorrhage, nothing to indicate basal fracture. However, let me state this very positively, that because there is an absence of these symptoms and a negative x-ray, basal fracture or fracture of the skull can by no means be ruled out. Speaking for a fracture, we have a very prominent man who says that following an injury to the head, a lucid interval, followed by coma, probably means only one thing and that is the hemorrhage of fracture. Usu-

ally we think of a depressed fracture, or comminuted fracture, but it may be nothing but a fissure and may be responsible for tremendous damage, not necessarily at the time, but later. Fracture can be easily fitted into this picture.

Upon admission a lumbar puncture was done and 5 c.c. of fluid removed. We believe that was done for diagnostic purposes, and we have the reading of the bloody contents and goldsol. The pressure was tremendous—400 millimeters of water and the normal only about 15. They knew she had a tremendous intracranial pressure; they knew that they had blood and serum in the cerebro-spinal fluid, and they knew she did not have meningitis or syphilis causing this condition. So, a few hours later she was doing so badly they decided to remove some of that pressure—a second puncture was done and 30 c.c. of fluid removed. Ten minutes later the patient died. Are we justified then, working in a crab-like fashion, in saying that because they did a lumbar puncture to relieve pressure, she did not have a fracture? No! Because with a fracture with intra-cranial pressure the most modern thuoght is that it should be removed in this manner. However, a pressure cone evidently formed following the lumbar puncture, allowing the pressure above to push down the medulla into the foramen magnum, thus causing paralysis of respiration.

We are making a diagnosis of hemorrhage—whether due to disease wholly and increasing pressure of cerebro-spinal fluid (with blood in it), or whether due to fracture, or a combination of the two, we are not prepared to say.

Discussion at Massachusetts General Hospital

RICHARD C. CABOT, M. D.

NOTES ON THE HISTORY

When patients come here in coma we are unusually prone to make mistakes in our diagnoses because we depend so much on an accurate history, which that particular condition precludes. We have very little given us up to the day of the onset.

"Since that morning she had not urinated." Of course we argue that that may be due (a) to accumulation of urine in the bladder due to coma or (b) to a failure of secretion. My guess is that it is the first.

We have really very little evidence here before we come to the physical examination. We have a high arterial tension. We have coma and apparently paralysis of one arm, which so often goes with coma. We may or we may not find something definite in the brain.

NOTES ON THE PHYSICAL EXAMINATION

"Percussion measurements not recorded." That was wise, I think.

"Fundi appeared normal." If that was a good examination that is important.

"Neck not stiff." They are looking for meningitis.

The reflexes are just the opposite of what we should expect. We generally get increased reflexes on the affected side. Again, the Kernig is on the wrong side.

That is a rather dubious, equivocal urinary sediment. It could go with a nephritis and perfectly well with a normal kidney.

The non-protein nitrogen is practically normal.

They were courageous men. They must have been perfectly sure there was not a brain tumor there, for they did a lumbar puncture. I have seen many deaths due to lumbar puncture in cases of brain tmor, the brain pressing down into the foramen magnum. They must have been very confident to exclude it. Some symptoms of it are present here. Puncture was done of course to relieve coma. It sometimes does, but it is not obvious to me that lumbar puncture did good.

DIFFERENTIAL DIAGNOSIS

We have blood in the spinal fluid and therefore presumably blood in the brain. Hence presumably the case is cerebral hemorrhage—cerebral hemorrhage in a person with hypertension, with presumably some enlargement of the heart, with presumably normal kidneys; and that is all that I can say. I hope we examined the brain, Dr. Mallory?

Dr. Tracy B. Mallory: Yes.

Dr. Cabot: I commit myself then. I say it is cerebral hemorrhage Which side it is on I am in doubt. I think it is probably on the left.

A Student: Do you think that the lumbar punctures had any effect in producing blood in the puncture fluid?

Dr. Cabot: I think it is safe to conjecture that they excluded this.

A Student: How can you be sure, if the patient struggled?

Dr. Cabot: You can not.

A Student: There is no record of a sugar test in the urine.

Dr. Cabot: That is true. I am glad you pointed it out. But I do not believe that she died of diabetes. Do you?

A Student: Do you thnk the last baby had anything to do with this? There are no findings in the viscera of toxemia.

Dr. Cabot: I think not. She was well during most of this month. If she had not had the cerebral hemorrhage she would have been well a long time more, I think.

A Student: The fundi were normal. Would not the lumbar puncture be therefore justified?

Dr. Cabot: That is a good point. The absence of edema in the retinae tends to exclude tumor and tends to make it safe to do a lumbar puncture, though it is not absolutely reliable. But I imagine it was done chiefly for diagnosis, which of course would be important if there was fever.

A Student: Do you consider falling down stairs immediately responsible for the lesion?

Dr. Cabot: No. I think it is the other way. Sometimes it is a question whether or not a person falls accidentally, strikes his head and gets a fracture or becomes dizzy from brain lesion and so fails to obstruct his fall. It is very hard to decide. But there is no fracture found by the x-ray.

A Student: Which came first, the hypertension or the cerebral hemorrhage?

Dr. Cabot: I think probably the hypertension.

A Student: Do you think she had more than one hemorrhage?

A Student: It is stated that the blood pressure before delivery was normal.

Dr. Cabot: That is rather a staggering fact. It is only a month since then. That is a short time to get up a high blood pressure. That rather tends to show that the hypertension is due to the cerebral hemorrhage rather than vice versa.

A Student: Why did they do the second lumbar puncture?

Dr. Cabot: I do not know. I suppose they thought that perhaps they did not get a fair specimen the first time. But as long as they got crenated red cells I think they got enough evidence.

A Student: What do the urinary findings mean? She had bloody urine, leukocytes ahd albumin.

Dr. Cabot: I should not say she had bloody urine. By that phrase we ordinarily mean gross blood that you can see with the naked eye. You see fully as much as this after a foot ball game in normal men, fully as much after a boat race. You see as much as this in pneumonia when the kidney does not show anything uost mortem.

A Student: According to this the specific gravity is fixed.

Dr. Cabot: High fixed specific gravity does not mean anything, so far as I know. It is perfectly conceivable that there is a nephritis, but I do not think that we have evidence of it. The pupils in coma are useless. I have seen a great many pupillary chnages in cases of coma and very seldom have found anything post mortem to explain them.

A Student: Are there not some things here suggesting syphilis?

Dr. Cabot: What?

A Student: Cerebral hemorrhage in a young person.

Dr. Cabot: Yes. She is young for cerebral hemorrhage.

A Student: Isn't that goldsol suspicious of luetic disease?

Dr. Cabot: I should not say so. Of course we can not with certainty exclude lues. We never can. We have a negative Wassermann, but that is not enough to exclude the disease. I think we have very little evidence of it aside from that point mentioned, that lues is a common cause of early cerebral hemorrhage.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Intracranial hemorrhage.

DR. RICHARD C. CABOT'S DIAGNOSIS

Hypertrophy and dilatation of the heart.
Hypertension.
Cerebral hemorrhage.

ANATOMIC DIAGNOSIS

1. *Primary disease.*
Cerebral hemorrhage.
Acute intracapillary glomerulonephritis. (Nephritis of pregnancy?)
2. *Secondary or terminal lesions.*
Focal hemorrhages in the liver, slight.
Subinvolution of the uterus.

DR. TRACY B. MALLORY

This was a puzzling case clinically, and it is still something of a puzzle from a pathologic point of view.

To start with the most obvious things, she did have a cerebral hemorrhage. In the left p arietal lobe was a large clot and surrounding softening of the brain tissue 9 centimeters in diameter. There was also a considerable degree of subdural and subpial hemorrhage scattered rather irregularly over the entire surface of the brain. That checks pretty well with the clinical findings, with the one definite symptom, paralysis of the left arm, and left parietal intracranial hemorrhage to go with that. The other reflex changes were rather vague and might easily have been caused by the subpial hemorrhage on the surface.

The heart weighed 350 grams. That is very slight enlargement for a fairly large woman. Consequently we must presume the hypertension was not of iong duration, and that agrees with the statement that the blood pressure was normal some time before her delivery.

The liver was considerably enlarged. In gross I was not able to make out anything abnormal about it. Histologically however we did find a few small focal hemorrhages and minute healed areas of focal necrosis.

The kidneys weighed 325 grams. They were very deep red in color. The markings were normal. There were no hemorrhages beneath the capsule. The cortex measured 8 millimeters, perhaps slightly increased. Microscopic examination showed an extremely acute intracapillary glomerulonephritis, every glomerulus being filled with polymorphonuclear leukocytes, 20, 30 and 40 of them in each glomerular cross section.

Now to try to put the thing together. The glomerulonephritis could have caused the hypertension, possibly could have caused the cerebral hemorrhage. A nephritis, focal lesions in the liver, and cerebral hemorrhage are a combination very suggestive of eclampsia. Nine weeks after delivery is a long time for eclampsia. I have seen it two weeks after delivery, but not later that that. What is your experience, Dr. Cabot?

Dr. Cabot: I never heard of it after so long a period.

Dr. Mallory: It certainly is not a typical case. Except for the long period—and I do not think that it is utterly impossible that eclampsia should be delayed as long as that—that would be the best explanation for the combination of the lesions.

Dr. Cabot: There was no special evidence of syphilis?

A Student: Was a blood culture done?

Dr. Mallory: There was a blood culture post mortem that was negative, but not during life.

A Student: What is the etiology of eclampsia?

Dr. Cabot: Nobody knows.

A Student: Did this woman come out of her coma before she died?

Dr. Mallory: No.

Dr. Cabot: There are various degrees of coma. One can answer questions and act reflexively, and think of course. It could not have been a very deep coma.

CASE REPORTS AND DISCUSSIONS FROM VALMORA SANATORIUM

C. H. GELLENTHIEN, M. D.
Valmora, N. M.

At the staff meeting of Valmora Sanatorium, held on Dec. 8, 1928, several interesting cases were presented and discussed. Those present were Drs. F. H. Crail of Las Vegas, H. W. Gibbs of Las Vegas, W. E. Kaser of Las Vegas, E. B. Shaw of Las Vegas, J. H. Steele of Wagon Mound, W. J. Latta of Wagon Mound, W. T. Brown and C. H. Gellenthein of Valmora. The cases discussed presented a variety of diagnostic and therapeutic problems:

The Valmora Sanatorium is maintained by a number of large Chicago corporations for their employees who break down with tuberculosis. Dr. W. T. Brown is superintendent and Dr. C. H. Gellenthein is medical director.

CASE NO. 1

In the first case there is still some question; it is hoped that the group here tonight will make some helpful suggestions. He came with a letter dated March 27, 1928, from Dr. Johnson of La Junta as follows:

"I am sending you who first

entered this hospital on January 3, 1927, with a collapsed lung. He was watched by x-ray and by repeated examinations, and this lung expanded within a month and remained so. Our pictures show an apparent cavity in the base of the lung. He improved so much in strength, that we allowed him to return to work, but he has again returned to us, with his sputum showing some blood, especially after exercise. Repeated examinations have been negative for tubercle bacilli, and there has been some suspicion that he has tuberculosis. If he has a malignancy, it is an unusual type. Recent x-ray is much clouded in both lungs. Previous pictures clear except at base."

Past History: Born in Wales, where he lived for 7 years; has lived in Pueblo, Colo. 42 years; worked in steel mills two years; has been blacksmith 27 years.

Family History: Negative; married 28 years; health of consort good; no known exposure to tuberculosis.

Personal History: General health fair during first five years; scarlatina at age of three; measles, mumps, pertussis in childhood; quinsy when child. Health in youth and early adult life good; vaccinated in 1905; typhoid in 1901; rheumatism in 1910; right ankle crushed in 1911, no anesthetic; poisoned by canned lobster in 1914; influenza in December, 1926, with pleural effusion. Gonorrhea and syphilis denied.

Present Symptoms: Paroxysmal, tight, productive cough, most severe in morning and on changing position. One-fourth cup expectoration in 24 hours, yellow, mucoid, blood-tinged. T. B. absent (Jan. 2, 1927). Dyspnea when lying down and on exertion; strength poor; fatigue easily. Appetite fair, digestion unimpaired, aversion to no foods, drinks 3 to 4 glasses of water in 24 hours. Urination 5 to 6 times in day, twice at night. Bowels regular; hemorrhoids. Sweats on exertion. Sleep normal on left side; feels rested in a.m. Burning pain under sternum; cold hands and feet. One cup coffee a meal; four cigarettes per day.

Physical Examination: Average weight last three years 125. Highest weight 145 in 1902; lowest was 115 in June, 1928. General condition, good development, fingers cyanotic nails curved, neurotic; reflexes negative. Lips pink; teeth all out; tongue moist and coated. Voice husky at times. Heart murmur. Liver normal. Chest long, broad and deep; clavicles and scapulae prominent; shoulders square; right handed; scoliosis with left deviation in lumbar region, flexible. Breathing mixed; lagging right excursion. Temp. 96 to 99; pulse 86 to 100; height 5 ft. 5 in.; weight 117 lbs. Depression above clavicles right and left. Atrophy of scapular muscles both sides.

Chest examination revealed no definite findings, and during his residence here we were unable to find anything but vague, indefinite evidence of lung involvement. There have been no rales.

We will now have tell his own story:

"December 22, 1927, I was working as blacksmith. Did not feel bad, but that evening went to wash hands and pain struck me through the right breast to the shoulder. I walked home and took 45 min. instead of 15 min. as it usually did. The sharp pain continued. Took warm bath and wife put on hot water bottle and Antiflogistine. In the morning it was the same, but next day better. Called the doctor, but he did not seem to understand it well. Stayed in bed for a few days and got up some thick clots of blood and my breath was very short. This continued for several days. Got up and walked around, but still short of breath. Went to doctor and he told me to go to bed. Gave me examination next day and ordered me to hospital at La Junta. Still got up blood. They x-rayed me and found out that my right lung was collapsed. From then on just coughed up this black blood. After being at hospital for 60 days, doctor gave permission to go home as I wasn't coughing up blood. Must have exerted myself for on return to hospital continued to get up blood. Kept me at hospital but not in bed. They were very much puzzled. Two doctors thought it was tuberculosis, but one did not. Conference at La Junta of doctors from Ft. Lyons and they sent me there for x-ray. Gave diagnosis of cancer of lung. I asked the doctor what he thought about my outlook and he said I had better call in the undertaker. I went home and so far it hasn't proved to be cancer of the lung. Was ordered to Valmora and Dr. Gellenthien has had me in charge ever since. Was in bed here for 10 or 12 days and coughed up black blood. Sent me to clinic at Pueblo Doctor there examined sputum for three weeks and came to conclusion it was not tuberculosis After the first five months went back to work and worked for four months apparently getting along fine and had slight hemorrhage of black blood. Sometimes the sputum is streaked. Other times it is just spotted."

At the time first x-ray was taken January 3, 1927 he was still expectorating. He complained that at first breathing intensified pain, but not now. Dyspnea at times. When coughing felt lump in right lung which raised up on coughing. Cough loose at times. Most severe in morning at first, not now. Spasms of coughing continue until vomiting. Sputum gray, streaked with blood. Fourth cupful in 24 hours. Oral rales present. Changing of position used to aggravate cough. In going over the x-rays, there are shadows at the base that have been diagnosed cavities, but we felt that they were not, but were examples of bullous emphysema. A second picture was taken February 9. 1927, shows thickening of bronchial tree extending up almost into apices. Diaphragm erect, narrow vertical heart. Absence of calcified nodes and consolidated lung tissues..

In the picture taken February 25, 1927, cavities at right base are smaller and walls of cavities left base not as definitely demarcated. Film on March 13, 1927, shows cavities at right base apparently merging and left base cavities almost disappeared. Films on March 29. Sept. 3 and Oct. 22. 1927, showed little further change. On January 14, 1928, film did not show much change; cavity right base gone, left base present. Cavities usually present no such behavior as this. On February 20, 1928, diagnosis of carcinoma was made at Ft. Lyons on x-ray film. On May 8, 1928, suspicious feathery streaking up into left apex. On July 13, 1928, greater extension of interlacing dense fibers into left apex. On September 4, 1928, interlacing stil evident into left apex, right cleared up.

In our analysis of this case we have a man 49 years old, family history negative. Onset ruptured right lung, etc. His temperature range now is from 99.2 to 95. Pulse 120 to 70. Not much difference in his symtoms. He has cough, expectoration blood streaked sputum. No night sweats nor chills. Has languor, anorexia, lack of endurance and strength.

Laboratory Reports: Sputum on December 5, 1928, gray, no odor, tenacious mucus; slightly alkaline; no elastic fibers, many epithelial cells, few pus cells. No tubercle bacilli. Other bacteria, staphylococci, streptococci and pneumococci. Guinea pig died of a septicemia 5 days after inoculation with some of the sputum.

Von Pirquet negative. Urine negative.

On admission, B.P. was 122/70, red count 4,520,-

000 and white count 5400. At present B.P. is 108/70, red count 4,830,000, hemoglobin 90, white count 6200.

The sputum has been watched for fragments of tumor mass with no avail. The question resolved itself to tuberculosis, carcinoma or bronchiectasis. And we finally concluded that it was a case of bronchiectasis. I showed the films to Dr. Sampson at Saranac Lake, and he confirmed my findings. Now we intend to make an intra-tracheal injection of lipiodol which should give us evidence of the dilation of the bronchi and definite fluid levels, with film taken in the vertical position. Bleeding is due to erosion of the bronchial wall and collection of blood in the sacs of the bronchi as in hemoptysis from a cavity.

Bronchiectasis per se is unusual. According to Lord, one-third of all cases are secondary to tuberculosis. The dilation of the bronchi may be cylindrical, fusiform or saccular and there are three theories /attempting to explain bronchiectasis. (1) Bronchial: By Laennec. A bronchial catarrh and stagnation of secretion with the mechanical effect of cough and a disturbance in the nutrition of the bronchial wall, with loss of elasticity, contractibility and ciliary motion. (2) Pulmonary: Advanced by Corrigan in the Dublin Journal 1838, and according to him, due to a shrinkage of fibrotic lung tissue between the bronchi. (3) Broncho-pulmonary: A combination of the pulmonary and bronchial theories.

Dr. Latta: I have seen clotting in gangrene and I wonder how you ruled out gangrene and lung abscess. I had one case of carcinoma of the gall bladder and liver with formation of biliary sinus draining into the lower right lung, some 20 years ago.

Dr. Gellenthien: If this were an abscess or a lung gangrene the sputum would be purulent and would have a very foul odor and the general symptoms wold be those of a septicemia with rigors, chills, high temperature, sweats, etc.

After we had committed ourselves to the diagnosis of bronchiectasis, went to Pueblo, where he was under Dr. Maynard's observation for three weeks. Following is his report:

"Specimens of sputum have been submitted on June 22, 23, 25, 30, July 3, 4, and 6. Specimen of July 4 was old when brought to laboratory, so was discarded.

Fresh slides of all specimens have showed pus and epithelial cells but no elastic fibers, spirals or crystals. On June 23, a few large spores were present, but no mycelium, and the spores have failed to grow on agar slants after 2 weeks at room temperature.

No tubercle bacilli have been found, even in sputum incubated for 24 hours at 37° C. Streptococci were abundant in all specimens. Specimens of June 25, 30 and 23 separated into three layers, on standing, with pus at bottom, liquid in center, and mucus on top.

The 10 a. m. specimen of June 30 contained a few large pigment-filled cells. The latter specimen the same day contained many of the same cells, and on July 3 they were very numerous. They are 25-35 microns in diameter, thickly packed with brown pigment, which stained blue when treated with potassium ferryocyanide solution and hydrochloric acid. The July specimen did not show any pigmented cells. No eosinophiles have been found in any samples.

The gross appearance of these samples suggests a bronchiectasis. The absence of tubercle bacilli is of only slight negative value. With the history of a collapsed lung at first, the presence of dark blood in grouped samples and of the many large cells containing blood pigment, suggests an atelectatic bronchiectasis, due to a disintegration of the airless portion of the lung.

I have found no evidence of a fungus infection, the few spores present in one sample probably being a contamination."

Dr. Kaser: It is a peculiar thing, with that complete collapse that he should still have lots of blood. A complete collapse would stop breathing.

Dr. Gellenthien: Why should it collapse down as it did, to the base, rather than at the hilum?

Dr. Kaser: You may have pleuritic adhesions.

Dr. Gellenthien: The bleeding undoubtedly came from a portion of the lung held out by the adhesions.

Dr. Kaser: You should have had a bronchiectasis in the beginning of this case before the spontaneous collapse occurred.

Dr. Crail: The first symptoms evidently were perforation of some portion of the bronchial tree. The common cause of spontaneous pneumothorax is tuberculosis. One other thing that causes it is influenza. While in the army during the war and while in Houston we had numerous cases of lung collapse due to influenza. This history is too long for carcinoma.

Dr. Shaw: The history preceding would indicate tuberculosis infection with its loss of weight and little cough.

Dr. Gellenthien: We should be getting physical examination evidence in the chest, but have not so far. The x-ray however, does show faint interlacing fibers extending almost to the periphery and the apex in the left lung.

CASE NO. 2.

Male, age 20, born in Chicago; parents born in Germany, but patient has lived entire life in United States. Family history irrelevant, without known exposure to tuberculosis. Vaccinated in 1915; has had bronchitis, measles, pertussis and pleurisy with effusion. Gonorrhea and syphilis denied.

Present illness: Onset in May, with cold. In August 1926 had hemorrhage (cupful), bed rest 7 weeks. Slight cough, night sweats. February, 1927, right lung collapsed (pneumothorax). Kept collapsed until June, 1927. January, 1928, re-collapsed. In April empyema developed. Aspirated for few weeks. Resected two ribs in April 1928.

Present Symptoms: Cough most severe during morning and after laughing; loose productive and paroxysmal. Expectoration yellow, green, gray, one drachm in 24 hours Micro. exam. June 1926, April 1928, T.B. present. Hemoptysis first August 1926, one cup full; last October 1927. Temporary hoarseness. Dyspnea on exertion. Strength, fair. Fatigue easily. Can walk about two blocks. Appetite poor. Digestion impaired, nausea and gas. Aversion to acids. Thirst normal, drinks 8 glasses in 24 hours. Urinates 5 to 6 times day, once at night. Bowels regular. Hectic time 4 a. m. Slightly flushed cheeks. Sleep normal 10 hours on back, rested in a. m. No chills; cold hands or feet during the day. Weight, highest 155 lbs. in March 1928. Average last 3 years 135. On admission 108. Coffee one cup. Tobacco in moderation two years ago.

Physical Examination: General condition good. Medium complexion. Fingers clubbed, cyanotic, curved nails, sweaty palms. Development good; anemia slight mucous membranes pale. Nervous system normal, pupils of eyes react to light and accommodation. Lips pale. Teeth fair. Tongue moist and coated. Liver normal. Long narrow shallow chest; clavicles prominent; scapulae prominent; drooping shoulders. Right handed. Mixed breathing. Respiratory marked lagging on right. Oral rales present. Atrophy of scapular muscles right and left. Left: Interlobar pleurisy.

Chest findings: Rales throughout right lung, front and back, and to the 3rd rib anteriorly and to the base posteriorly in the left lung. Cavity at right apex, distant breathing right base.

Diagnosis: Far advanced pulmonary tuberculosis: Prognosis unfavorable.

About June, 1926, patient entered hospital in Chicago, was x-rayed and has positive diagnosis of pulmonary tuberculosis. On Aug. first or second he had hemorrhage of cupful in amount, after which he was kept in bed seven weeks and was then up on exercise. Had but slight cough at that time. Although apparently doing all right, he was x-rayed in January 1927, showing excavation at right apex. His doctor started pneumothorax on Feb. 1, 1927, inserting gas every two or three weeks. He gained weight, cough was much better and he improved in all respects. X-ray on Feb. 5th showed apex not collapsed; on March 3rd collapse was slightly less; on April 15 fluid was shown to be forming; on June first fluid had increased. On June 15th pneumothorax was stopped and he was put on bed rest. On July 22, x-ray showed lung creeping out in the fluid; on Sept. 2nd collapse had about disappeared; on Nov. 17 fluid had decreased. On Jan. 12, 1928 he was re-collapsed, following which he had much pain in chest and his doctor removed some fluid which was yellow and clear.

At end of March sick at stomach, had night sweats but no chills. April 18, 1928, had severe pain on sitting up. Diagnosis of empyema made with puncture needle. Removed pus every week with the needle from April 15 to May 10.

May 12, 1928. Rib re-section was done under local, with immediate relief. June 1, 1928 went home, night sweats gone. Wound was left alone until July 15, 1928. Then irrigated once daily with Dakin's solution until coming here, October, 1928.

Laboratory Report: Sputum: December 5, 1928. Yellow, green, gray; foul odor; muco-purulent; alkalin reaction; numerous elastic fibers; few epithelial cells, many pus cells. Positive for tubercle bacilli (Gaffky 8), forms beaded; few staphylococci, and few pneumococci.

Urine: Negative.

Blood, December 8, 1928: Wassermann, negative. B.P. 118/668; hemoglobin 90; erythrocytes 4,380,000; leucocytes 9,000.

Temperature range now 100.8 to 97.0. Pulse 122 to 92. Weight on admission 108 lbs., now 112. During the past 8 weeks, cough has improved slightly. Appetite improved, digestion good, bowels regular. He has had no night nor day sweats while here. No pain except pleurisy at left base. Strength is improved. The drainage is nearly the same, seems slightly better. Did not have a tube when he came and it has not been necessary. Smears made from the secretion have all been negative.

Dr. Crail: This would seem to be an ideal case for thorocoplasty. His left lung seems to be in fair condition and the load of toxemia would be much less, if the right were collapsed.

Dr. Gellenthein: That is true, but at present he is responding well to rest. His symptoms are decreasing and his weight is increasing.

Dr. Shaw: Does air pass in and out through the sinus opening?

Dr. Gellenthien: At first it did, but not at present. We are having no difficulty in getting proper and sufficent drainage.

Dr. Crail: Is there anything in the left lung except pleurisy at left base?

Dr. Gellenthien: Rales throughout back in the left lung.

Dr. Crail: What do you think his prognosis in that case is?

Dr. Gellenthien: Not so good.

Dr. Latta: Were there enough ribs taken out?

Dr. Gellenthien: The surgeon had no intention of effecting a lung collapse, and enough ribs were re-sected to give ample drainage. We want to put him on heliotherapy in the spring and it might close that sinus. Last picture indicated a great deal of scar tissue.

Dr. Gibbs: Would phrenic exeresis bring diaphragm high enough to fill that space?

Dr. Gellenthien: Do not think so; it would bring it up some, but not enough.

Dr. Shaw: How do you give sun treatment?

Dr. Gellentheien: Sun in pulmonary tuberculosis is a difficult hting. It general we don't know what we are using, and what its effects will be. For all practical purposes we are using two rays, the ultra-violet and the infrared. The ultra-violet is the more potent ray, and its reaction is general or systemic; we have no fear of exposing the entire body to it with our lamps. The infrared ray is the heat ray and is local in its reaction; if the chest is exposed to it injudiciously a pulmonary congestion with a resultng hemorrhage may occur. At Valmora the dosage is controlled by Rollier's technique and actinotherapy is not used in an acute pulmonary tuberculosis. When the case becomes quiescent sun is given as exercise. So far we have had no ill effects result from the use of the lamp or sun, and feel that we have gotten worth while benefits from their use. Rollier claims that one-third of actinotherapy treatment is due to the light, one-third is due to it psychic effect, and one-third due to other unknown factors.

Dr. Shaw: It would seem to me that if its only effect were psyhic, that it would still be an important worth while adjunct to treatment.

Dr. Gellenthien: Unquestionably so, and we know that the ultra-violet ray is almost a specific for intesinal and bone tuberculosis.

CASE NO. 3.

Unmarried female, age 30. Has lived in Topeka, Kans. ten years. No known exposure to tuberculosis.

General health during infancy good, fair during childhood, good during youth. Had mumps, pertussis and measles in childhood; typhoid in 1907; pleurisy with effusion in 1910. Ulna fractured in 1917; surgical operation for periostitis in 1917, with sequestrum removed in 1918. Diphtheria in 1918. Appendectomy under ether in 1919. Tonsillitis frequently with tonsillectomy in 1923 under local. Acute indigestion in 1927. Rectal fistula, fissure abscess under local in 1928, at Mayo Clinic. Menstruation began at age of 16 and has been normal since.

Present Illness: Onset December, 1924; in bed for 4 to 5 weeks with pleurisy at base. Never has left since. Slight cough, no night sweats, chills. Slight loss of weight. No hemorrhage, tired all the time. Went to Albuquerque May, 1925, for 4 months. Worked in Topeka for 2 years. January, 1927, back to Albuquerque for 6 months. Worked in Topeka for 13 months until coming here. Albuquerque Sanatorium first time 2 months, second time 6 months.

Present Symptoms: Cough most severe during morning. Character loose, productive. Expectoration yellow, blood tinged a very few times; amount in 24 hours 2 drachms; Micro. exam. April, 1927, T. B. absent, blood tinged 3 or 4 times in the last year. Loss of voice winter 1927. Strength poor, fatigue easily. Good appetite. Digestion unimpaired. Slight gas. Aversion to fats. Normal thirst; drinks 5 glasses. Urinates 3 to 4 times durng day, none at night. Bowels constipated. Hectic (time) 5 p. m. cheeks flush, head-

ache. Sleep good 8 hours on right side, not rested in a.m. Menstruation 25 to 26 days, normal flow, no pain. Pleurisy severe at right base. Cold feet. Weight: Highest 140½ August 1927, average last 3 years 132. Lowest recent 120, June 1928. Coffee 1 cup, tea 1 cup.

Physical Examination: General condition good. Development good. Nervous system normal. Pupils react to light and accommodation. Reflexes negative. Lips pale. Teeth good. Tongue moist, coated. Tonsils out. Abdomen, appendectomy scar. Liver normal. Long narrow shallow chest. Clavicles prominent. Scapulae prominent. Shoulders drooping. Right handed. Spine straight, flexible. Breathing abdominal, lagging right. Pulse 80. Respiration 32. Inspiration 33¼. Expiration 31¼. Breath 32. Depression above clavicles right and left. Atrophy of scapular muscles right and left.

The day before Thanksgiving she had a severe attack of neuritis and we had to give her morphine for relief. Severe pleurisy has started again. We did a Von Pirquet yesterday. As you see it is negative at the end of the first 24 hours. The 40 and 72 hour readings will probably be the same. Wassermann is negative.

Laboratory Report: Sputum December 5, 1928. Color grayish. Odor none, consistancy mucoid. Slightly alkaline reaction. Few elastic fibers, few epithelial, many pus cells, no T. B. Other bacteria staphylococci, streptococci pneumococci.

Urine: December 6, 1928. Slightly turbid; light amber; sp. gr. 1.012; aromatic odor; acid; no albumin nor sugar, bile nor hemoglobin; few pus cells; phosphates present; no acetone; slight trace of indican; no reaction; sediment, many epithelial cells, few uric acid crystals.

Blood: December 7, 1928. B.P. 120/90; hemoglobin 90, erythrocytes, 4,720,000; leucocytes 7,600. 7,600.

Dr. Gellenthien: With the fluoroscope nothing was evident, except that there were definite spasms of the right hemidiaphragm. Her temperature is normal in the morning. Starts at 96.8 goes to 100. Her pulse is from 92 to 70: With bed resst she has gained weight, but coughs more and temperature is higher than it was.

Dr. Shaw: The fact that one fails to find T.B. is not proof that there are none.

Dr. Gellenthien: According to Brown, at least one of five things are necessary to make a diagnosis of clinical tuberculosis (1) persistent rales in the upper half of the chest, (2) a parenchymatous x-ray lesion; (3) tubercle bacilli in the sputum; (4) history or evidence of wet pleurisy or (5) hemoptysis of a drachm or more.

Dr. Crail: Have you found any rales in this case?

Dr. Gellenthien: None.

Dr. Kaser: I found nothing upon physical examination. May I raise the question as to whether the response to tuberculin and the Von Pirquet disappear in some cases.

Dr. Gellenthein: At the recent Southwestern Medical meeting at Albuquerque I asked Dr. J. I. Myers of Minneapolis, whether the immunity or so-called allergic response of an individual disappeared as the lesion healed, especially in juvenile tuberculosis, where every symptom and sign disappears, even with the x-ray. He stated that in a large percentage allergy does not disappear, but in some it does. Some writers claim that the allergic state is lost in pregnancy and in far advanced terminal cases.

Dr. Latta: Muscle tenderness is a sign of tuberculosis and I find it in this case.

Dr. Shaw: In this case your signs are lacking, but symptoms are numerous. Everything is there that makes up a tuberculosis case. It is difficult to explain pleurisy on any other basis and I think this tuberculosis.

Dr. Latta: How about hemoglobin?

Dr. Gellenthien: Ninety per cent. How about the elastic fibers in the sputum, do they indicate activity?

Dr. Gibbs: How many patients do you have with nasal mouth breathng at night or with snus trouble?

Dr. Gellenthien: I don't know, off hand.

Dr. Gibbs: Are there ill effects from tonsillectomy?

Dr. Gellenthien: Dr. Crail has performed major operations on three quiescent cases during the last year with no ill effects. In each of these cases there was marked improvement after the operation.

Dr. Gibbs: Will a tonsillectomy bring on tuberculosis?

Dr. Crail: I don't see why any operation should increase the disease if the thing the patient is being operated on for gives him a handicap. These cases that break down after a tonsillectomy are probably breaking down before the operation is done.

Dr. Gibbs: It is a question worth while. They say it only has ill effects when the tonsils are not entirely removed.

CASE NO. 4.

The next case we wish to present iss one of pulmonary tuberculosis in a ten-months-old infant. The father was diagnosed as moderately advanced pulmonary tuberculosis and admitted to Valmora, August, 1928. His sputum has been persistently negative. His baby was 6 months old, and we advised him to have it examined by a competent pediatrician. The baby had no temperature, pulse, night sweats, cough nor symptoms of any sort. It had its normal weight and apparently nothing was wrong with it. Physical examination revealed nothing except one pea-sized hard cervical gland on the left. The Von Pirquet was positive and the x-ray shows extensive bilateral involvement.

Dr. Shaw: Was the mother well?

Dr. Gellenthien: Mother's physical examination and x-rays were negative.

Dr. Gibbs: Did the baby gain weight normally?

Dr. Gellenthien: The baby was apparently healthy, being symptom free and gaining weight as a normal baby should. We have been treating the baby with the usual anti-tuberculus regime and stressing heliotherapy. When it was ten months old it was apparently normal, being symptom free and weighing 24 pounds. Juvenile tuberculosis nearly always gets well, with proper care and we feel the prognosis in this case is good.

CASE NO. 5.

Our next case came with the following letter from his doctor:

"He came in with the usual symptoms of a chronic wasting disease, listlessness, loss in weight, tired feeling and moderate sweating at night. He was coughing very little when he came here and raising but little material. I put him on expectorant and in the following days made repeated acidfast stains of his sputum, but was never able to find any tuberculosis organisms. Methylene blue stains showed numerous colonies of diplococci and other coccus organisms. In fluoroscoping his lungs we could see that his lungs were more or less involved throughout with some sort of progressive condition."

Physical examination revealed an extensive bilateral involvement of the lungs.

Wassermann: Negative. Sputum: Negative except once. We were never able to repeat this and I feel that we had a contamination with a hay or smegma bacillus. On admission he complained of a smothering sensation and a feeling of oppression inside of his chest. His sputum was blood streaked. When he ate he would gurgle a good deal, so much so that we were never able to let him go to the dining room and after he finished his meal he would re-gurge several mouthfuls. The pupil of his right eye was dilated, the left was normal and he complained of a unilateral ssweating of the right side of the head.

We felt that the bilateral extensive involvement in this case was not due to pulmonary tuberculosis, but was probably a secondary acute process and the fact that we had a man in the 60's made us want to try and rule out carcinoma. On eating he complained of a fullness in the chest, vomiting after eating, a sense of weight back of the sternum, dyspnea and difficult deglutition and raising a blood streaked sputum. After the acute process in his lungs had subsided we attempted to aspirate the stomach contents with a Rehfuss duodenal and stomach tubes. We encountered difficulty, however, and were unable to pass an obstruction in the esophagus just at the upper border of the sternum. We then fed him a barium meal and watched it go down under the fluoroscope. At the level of the cricoid cartilage a bulge or outpouching appeared. We made a picture of it and found it to be the size of a hen's egg. We made a diagnosis of a pulsion diverticulum of the esophagus at the level of the cricoid cartilage. This pouch was infected and the acute secondary pulmonary infection of the lungs was by extension. After this acute infection had passed off the signs of pulmonary tuberculosis were evident.

The treatment as given in the texts consists of first surgical, second, an attempt at keeping the diverticulum empty by manual manipulation and hygiene. In this case we got results by the latter. He is now back at work and feeling fine.

BOOK REVIEWS

CORRECTIVE PHYSICAL EDUCATION FOR GROUPS. By Charles L. Lowman, M.D., F.A.C.S., Attending Orthopedic Surgeon, Methodist Hospital of Los Angeles; Chief of Staff, Orthopedic Hospital School, Los Angeles. 500 pages, 99 illus., price $1.50. A. S. Barness & Co., New York City.

The men of our profession are every day meeting with deformities or potential deformities due to some malaligrment. In children these simple distortions are cast aside for nature's correction. Orthopedic surgeons and physical educators have proved conclusively that these children do not outgrow their flat pronated feet, knock knees, bow legs, etc.

The physician who has these problems confronting him will find comfort in this book. Perhaps the chapters of most interest to the physician in part one are those dealing with: The Influence of Posture on Growth and Development, Technic of Examination, Lateral Posture Deviation and Growth Divergencies and Applied Principles of Exercise. The proper classifications of children into groups and the equipment for schools are discussed clearly in part two. The physical educator will find great help in part four. Every phase of physical education may be found in this book and it is as helpful to the physician as it is to the physical educator. The book is divided into five parts and is beautifully illustrated. It has a good bibliography and no index could be more complete.

PROBLEMS IN SURGERY: University of Washington Graduate Medical Lectures for 1927. By George W. Crile, M.D., edited by Amy F. Rowland. Octavo volume of 171 pages, illustrated. Philadelphia and London: W. B. Saunders Company, 1928. Cloth, $4.00 net.

The medical public is so thoroughly cognizant of the work of George W. Crile that it would seem superfluous for the reviewer to make more than mere mention of the field of the book.

There are 17 pages devoted to the management of the acute infections. There are 10 pages devoted to a general consideration of the treatment of premalignant and malicious conditions. There are 36 pages devoted to operations on the bad-risk patient. There are 24 pages on the mechanism of hyperthyroidism. There are 20 pages on the diagnosis and operative clinic. There are 36 pages on bipolar interpretation of certain normal and pathological conditions.

The book is profusely illustrated with excellent pictures. The type is easy to read and the paper is of the best. There are 171 pages. There is no index.

BRONCHIAL ASTHMA—Its Diagnosis and Treatment, by Harry L. Alexander, A.B., M.D., Associate Professor of Medicine in the Washington University Medical School, St. Louis, Mo.; Associate Physician to the Barnes Hospital, St. Louis, Mo.; illustrated; Lea and Febiger, Philadelphia; 1928.

This is a small book of 171 pages. It is written primarily, it would seem, to expound the author's view of vagatonia to explain asthma.

The author is dogmatic in his statements. In his preface he states that asthma is an explanation of human hyper-sensitiveness. While this may be the true explanation, there are many who feel that this is no talways the case, or at least that other factors, as well as hyper-sensitiveness, play important parts.

The subjject, however, is well and generally treated. The bibliography is excellent. It contains in the neighborhood of three hundred and twenty names. There are many of these names, also, which have repeated references to contributions.

No new methods of treatment are advanced, other than the use of ephedrin and and the surgical removal of the superior, middle, and inferior cervical sympathetic ganglia.

The author is pretty careless in his quotations at times, as for example, on page 45, he referred to my theory as a "non-passive respiration," whereas it should be "non-passive expiration," and says that "forced expiration causes pulmonic vascular engorgement," whereas forceful expiration produces vascular engorgement within the bronchi only and the reverse condition of the pulmonic vessels. This indicates very clearly that the author has not taken the trouble to understand other men's views.

The publisher's work is of the highest type. There are a few mis-spelled words as for example, on page 30 mucous in the fifth line, used as an adjective, has the letter "o" omitted. On page 38 the third line of the last paragraph, the letter "r" is inverted.

If the reader will take these few minor criticisms not too seriously, he will find the book an excellent dissertation on the subject of asthma and will gain an understanding of asthma from its reading.

EL PASO COUNTY NEWS

THE EL PASO COUNTY MEDICAL SOCIETY were hosts at a dinner on February 11, given in honor of Dr. Chas. Scudder of Boston, Dr. Burton Lee of New York, Dr. W. B. Thorning of Houston, Dr. D. B. McEachern representing the American College of Surgeons, and Fr. C. R. Moulinier of the Catholic Hospital Association. Dr. Scudder gave a brief discussion of the modern treatment of fractures. Dr. Lee talked on the management of cancer. He also explained a clinical method of grading cancer which is used in his clinic. Dr. McEachern spoke of the necessity of hospital standardization. He appealed to the society to insist that the county build a modern hospital. Fr. Moulinier also appealed for a new county hospital to take the place of the present inadequate one. A large representation from the county society and from William Beaumont Hospital attended the dinner.

DR. LESLIE M. SMITH attended a meeting of the Texas Dermatological Society which met March 2, at San Antonio. The meeting was attended by fourteen dermatologists practicing in Texas. The meeting was devoted largely to the presentation and discussion of clinical cases. Dr. B. F. Stout of San Antonio addressed the society on "The Application of the Bacteriophage in Dermatology."

THE SOUTHERN BAPTIST SANATORIUM celebrated the tenth anniversity of its founding during March. A campaign is being conducted for an endownment fund to provide treatment for indigent tuberculous patients.

PERSONALS AND NEWS ITEMS FOR ARIZONA

DR. VICTOR RANDOLPH, of Phoenix, with Mrs. Randolph and family, left the latter part of March for post-graduate work in Europe; he will be gone six months or longer, and will devote his attention chiefly to the surgical treatment of tuberculosis and other chest conditions, and to the study of heart diseases.

DR. GEORGE BRIDGE, formerly chief surgeon of the Copper Queen Hospital, in Bisbee, has resigned from that work and has moved to Phoenix. He expects to enter the practice of surgery in Phoenix some time this fall or winter.

DR. ROBERT FERGUSON, of Bisbee, who has been with the Copper Queen Hospital for many years, has been made chief surgeon in place of Dr. George Bridge, resigned. The Phelps-Dodge Company is constructing a new hospital in Bisbee which is said to be the last word in hospital design for industrial work.

DR. NORMAN ROSS, of Phoenix, has resigned as resident physician at St. Joseph's Hospital, and entered into practice in association with Dr. A. M. Tuthill.

Dr. CHARLES W. SULT, of Phoenix, has returned to his work, after an illness of several weeks from pneumonia.

DR. JAMES VANCE, of El Paso, was among the contestants in the recent southwestern golf tournament in Phoenix. With DR. KIM. BANNISTER, of Phoenix, he went to the semi-finals. Both the doctors suffered defeat, although putting up game fights.

DR. ROBERT J. DOSTAL, formerly with the United Verde Hospital in Jerome, has moved to Santa Monica, Calif., where he will engage in the practice of pedistrics

DR. CHARLES W. BROWN, of Florence, Ariz., has placed his membership with the Maricopa County Medical Society. Dr. Brown is a graduate of Emory University, Georgia, and came to Florence last year, to take the place of Dr. Fred Perkins as physician for the Arizona State Prison.

YAVAPAI COUNTY (Ariz.) MEDICAL SOCIETY

YAVAPAI County (Ariz.) Medical Society 10 bf

The Yavapai County Medical Society and Medical Officers of Fort Whipple, hold their annual joint banquet on Tuesday evening, March 26th. This event celebrated the close of their postgraduate study course for the year, in which they presented their diagnostic studies on the Case Histories of Massachusetts General Hospital, which have been printed in this journal.

Group II was declared the winner of the contest, with a percentage of 97.17; Group I, with a grade of 93.91 was second, and Group III with a record of 93.5 was third. The members of the winning group were Drs. Southworth, Flinn (J.W.), Flinn (Robert S.), Devine, Sullivan, Thomas, McWhirt, Bassett, Hazel, Matschke, Linn.

The judges for the year were Drs. Herrick, Buck and Rene.

The average attendance for all groups for the year was 97.8 per cent.

C. E. YOUNT, Secretary.

PRESENT DAY WORMS ARE WISE

(F. C. GOODWIN)

(Letter submitted by Dr. C. E. Yount, of Prescott, as evidence that Yavapai County worms are not disturbed by anything as weak as turpentine.—Ed.)

Prescott, Arizona,
Jan. 23, '29.

Dr. L.—Dear Dr. will you please look Peter over and see if he has worms. If so give him some medicine to get rid of them. The nurse sent him home from school said he had worms. And he went to Dr. X 3 times that where she sent him. And Dr. X said worms were all gone and he wouldn't need to come to him anny more. so Peter went to school monday and the nurse sent him home again. saying the worms were not gone. Now Dr. you have something there that will chase them worms and please give Peter something for them. He has been out of school 2 weeks trying to get rid of a worm. I don't know if that nurse knows what a worm is I doubt it. I started to give him turpentine on sugar. that what they used to give me. But that was all most 50 years ago and things have changed so since then and every one got so wise. I was afraid maby the worms were wise too and wouldn't be fooled with a little turpentine. Give him something for that worm will you please Dr.

I am Respct. Mrs. ———

GRASSES THAT GROW

The care with which our advertisers prepare copy is shown by the Arlington Chemical Company in their page advertisement in this issue. The illustrations of grasses are prepared by the Artist Mary E. Eaton, being painted in the original pictures with the colors of the grasses. The Arlington Chemical Company has reproduced the colors in their literature Hay fever patients especially will be interested to see the actual pictures of the pollen grasses which cause them so much annoyance.

Southwestern Medicine

Printed by THE A C TAYLOR PRINTING CO, Phoenix, Arizona
Published monthly for the Board of Managers of the four constituent societies.

Volume XIII. APRIL, 1929 No. 4

EDITORIAL STAFF

DR. WARNER WATKINS, Box 1587, Phoenix, Arizona ..... Editor in Chief
DR. LESLIE M. SMITH, El Paso, Texas ..... Associate Editor
DR. ORVILLE H. BROWN, Phoenix, Arizona ..... Associate Editor
DR. L. B. COHENOUR, Albuquerque, N. M. ..... Associate Editor

BOARD OF MANAGERS

ARIZONA STATE MEDICAL ASSOCIATION

DR. D. F. HARBRIDGE ..... Phoenix
A. C. CARLSON ..... Jerome

EL PASO COUNTY MEDICAL SOCIETY

DR. JAMES VANCE (Chairman) ..... El Paso
DR. W. L. BROWN ..... El Paso

NEW MEXICO STATE MEDICAL SOCIETY

DR. C. F. BEESON ..... Roswell
DR. H. A. MILLER ..... Clovis

MEDICAL & SURGICAL ASSOCIATION OF THE SOUTHWEST

DR. HUGH CROUSE ..... El Paso
DR. W. WARNER WATKINS ..... Phoenix

NEW MEXICO MEDICAL SOCIETY

Taos—June 12, 13 and 14.

The New Mexico Medical Society, through Dr. L. B. Cohenour of Albuquerque, announce a very pleasing program for their annual meeting, which will be held this year at the famous Indian pueblo town of Taos, in the northern portion of the state. The tentative program includes the following speakers and subjects:

Dr. Wm. P. McCrossin, Colorado Springs—"Endocervicitis."

Dr. M. S. McGregory, Oklahoma City—"The Treatment of the Neuroses."

Dr. Wm. Senger, Pueblo, Colo.—"The Neglected Perforated Appendix."

Dr. G. Werley, El Paso—"The Management of Heart Failure and Dropsy."

Dr. Wm. M. Spitzer, Denver—"The End Results of Prostatectomy."

Dr. W. E. Sistrunk, Rochester, Minn., (Subject not announced).

Dr. W. Warner Watkins, Phoenix—"Fungus Infection with Special Reference to the Lungs."

Dr. Kelvin D. Lynch, El Paso—"Polycystic Kidney."

Dr. Frank C. Goodwin, El Paso, (Subject not announced).

Dr. Carl Mulky, Albuquerque—"Hodgkin's Disease with Case Report."

Dr. M. P. Beam, Albuquerque—"Cerebrospinal Meningitis, with Two Case Reports."

Dr. Leon Rosenwald, Kansas City—"Chancroid's and Their Specific Treatment."

Dr. W. L. Brown, El Paso, (Subject not announced).

Dr. Casper F. Henger, Denver—"Surgery of Pulmonary Tuberculosis."

Dr. Carl A. Meninger, Topeka—"Encephalography."

The excellent program, and the unique surroundings of this meeting should attract a large attendance. A vacation trip can be extended from Taos into other parts of New Mexico or into the Mesa Verde National Park and other parts of Colorado.

The officers of the New Mexico Medical Society are as follows:

President, Dr. C. B. Elliott, Raton.

President-elect, Dr. T. P. Martin, Taos.

Vice President, Dr. F. D. Vickers, Deming.

Secretary-Treasurer, Dr. L. B. Cohenour, Albuquerque.

Councilors: Dr. W. T. Joyner, Roswell.
Dr. H. A. Miller, Clovis.
Dr. W. E. Kaser, Las Vegas.
Dr. R. O. Brown, Santa Fe.
Dr. F. D. Vickers, Deming.
Dr. Carl Mulky, Albuquerque.

THE CIGARETTE PROPAGANDA

This journal, in common with the Journal of the American Medical Association and all other medical journals whose advertising pages are open only to copy approved by the Cooperative Medical Advertising Bureau, has refused the ads of the American Tobacco Company, on the grounds that their statements are untruthful and inimical to good health.

That phase of the advertising which has provoked a defense reaction from the medical profession is the attempt to capitalize the present desire for slimness in physique by advertising the use of cigarettes as a substitute for the natural appetite for sweets. As the Journal of the American Medical Association states: "the human appetite is a delicate mechanism, and the attempt to urge that it be aborted or destroyed by the regular use of tobacco is essen-

tially vicious." After all is said, the effect of the cigarette is a drug effect, and the advertising of the drugging of a natural appetite in a healthy person, is certainly vicious. We, therefore, commend to all good citizens, support of the movement to have the Federal Radio Commission ban this propaganda from the air, and the Federal Trade Commission to have these advtrtisements refused admission to the mails.

OUR DIAGNOSTIC OPEN FORUM TO CONTINUE

Although this plan has not yet won the interest it deserves from the county medical societies over the southwest, the Arizona State Medical Association, at the meeting in Prescott recently, authorized the expense which will permit the reporting of the Yavapai County discussions, and SOUTHWESTERN MEDICINE will continue to publish these as a worthy example for other societies to follow, or attempt to improve upon. In the New England Journal of Medicine for March 28, 1929, the various methods of using these case histories in clinical study, as developed by groups over the country, are described, and the following almost flattering comments on the "Yavapai County Plan" and our "Open Forum" are made:

An outgrowth of the work of the Yavapai County society has been developing during the past year with promise of a future of increasing value to medical men in the Southwest. In March 1928, *Southwestern Medicine*, the official organ of the medical societies of Arizona, New Mexico, El Paso County of Texas, and of the Medical and Surgical Association of the Southwest, began publishing the discussions of the Yavapai County and Fort Whipple group. By May the idea had developed to include other societies. With the co-operation of the Arizona State Medical Society it was planned to publish the clinical histories of the cases discussed by the Yavapai County Society without the discussions, inviting other societies to discuss them and send in their results for publication in *Southwestern Medicine* together with the discussions at Prescott and the Massachusetts General Hospital. By November the new "Open Forum in Diagnosis" had grown into a department of nineteen pages. In January *Southwestern Medicine* announced a further development. "There is now being offered by this journal a plan where by the smallest county society (we have one in Arizona with three members) can have regular and profitable meetings and keep itself on record. This journal will send to any county society printed copies of case histories sufficient to supply all their members. The members of that society can arrange to meet once a month at luncheon, at some member's home or at a centrally located office or wherever they choose, briefly discuss these histories, endorse their diagnostic conclusions on the history sheet and mail them back to this journal. They will then go on record as having held a regular county society meeting, with some valuable clinical discussions." Dr. Watkins, the editor, writes, "We are using the cases which are to be studied by the Yavapai County Medical Society. We then gather in such discussions of them as have been made at various societies and print the whole record,—(1) the case history, (2) discussions by various societies or individuals, (3) the discussion at the Massachusetts General Hospital, with autopsy findings." In this way *Southwestern Medicine* avoids the lapse of a month or two between the appearance of the head of a Case Record and its body, besides adding convenience and incentive to the discussion of these cases by county societies throughout the states in its territory. The whole vigorously developing plan is more than a notable feat of medical journalism; it is a genuine stroke of medical statesmanship.

MARICOPA COUNTY (Ariz.) MEDICAL SOCIETY

The Maricopa County Medical Society met Wednesday evening, March 27, for its regular meeting which was postponed from Monday evening, March 25, in order to have the privilege of having with us Doctors Burton Hazeltine and Alvin La Forge of Chicago, and Dean W. Myers of Ann Arbor, Michigan.

The following members of the Society were in attendance: Mills, Milloy, F. W. Brown, Shelley, Phillips, Wilson, Mason, Drane, Wheeler, Harbridge, Reese, Kennedy, McIntyre, Felch, H. Randolph, Franklin, McLoone, Greer, Bakes, Carson, Robb, Thayer, Wilkinson, McDonald, Schwartz, Armbruster, Holmes, Hamer, Browning, Bailey and O. H. Brown.

The president, Dr. H. T. Bailey, called the meeting to order. He appointed the delegates to the state meeting, which will be held next month, as follows: Drs. Wylie, Stroud, Watkins, Greer, Bailey, Franklin, McIntyre, and Goss; alternates, Shelley, Milloy, O. H. Brown, H. Randolph and McLoone.

He next brought up the question of the radio programs and commented briefly upon them. He suggested that the present incumbent of the office should continue in charge of the programs, but that he should have the assistance of any or all the members of the society wheen needed. Dr. Carson moved that Dr. Brown be continued as chairman of the committee and that he should feel free to call on the membership of the society for assistance. This was seconded and duty carried.

The secretary then reported that Doctors Howell Randolph and Charles W. Brown of Florence, had been accepted by the Board of Censors for membership in the Maricopa County Medical Society, and on the motion of Dr. Carson, seconded by Dr. Phillips, they were duly elected to membership in the society.

The secretary then reported that he had a communication from Dr. Watkins which, in brief, suggested that of the four medical meetings held each month in Phoenix—two staff meetings and two county society meetings—two be eliminated and that the time of half of each staff meeting be given to the county society or vice versa. On the motion of Dr. Wilkinson, seconded by Dr. McLoone, and carried, the chair was instructed to appoint a committee to work out the details of the proposition and report back to the society at the next meeting. The president appointed the presidents and secretaries of the staffs to work with the president and secretary of the county society.

The following program was presented by the visitors from the east.

The Clinical Problem of Bronchial Asthma

The General Problem; Burton Hazeltine, M. D., F. A. C. S., Chicago. The Internist's Problem; A. W. La Forge, A.M., M.D., Chicago.

The Rhinologist's Problem; Dean W. Myers, M. D., F.A.C.S., Ann Arbor, Michigan.

General Discussion.

Dr. Hazeltine led off and was followed by Dr. La Forge after which Dr. Myers gave his motion picture and lecture. Doctor Myers' paper was illustrated with a moving picture film showing technic of the rhinological procedures.

The gist of the remarks of the distinguished guests was to the effect that asthma is a chronic toxemia which produces reflex bronchial spasm. There are areas in the nose, above and posterior to the far end of the middle turbinate, which they designate as trigger areas because they are particularly prone to set up bronchial spasm when they are irritated. The reflex may be set up, however, by irritation at any point of the respiratory tract.

The patient should be thoroughly studied from all angles and standpoints, just as though ill with any other chronic condition, to discover all possible sources of toxemia. They say that it is entirely unnecessary to make allergic studies or tests. They record these for the purpose of satisfying the patient but for no other purpose as they regard them as thoroughly useless.

The patient, however, should be carefully studied so as to discover every possible focus or site which might be the cause of toxic conditions. The internist and the rhinologist are probably the two most important men in the study of a case of asthma. They should work hand in hand. Any other specialist needed to make an exhaustive study of the case should be employed. After all of the pathology has been discovered a program of cure should be agreed upon. They plan to treat first that area or pathologic focus which gives promise of giving the patient the greatest amount of quick relief. They take no case whch will not agree to follow out the plan of treatment as outlined for whatever length of time necessary. The nasal treatment is carried out with the most scrupulous care. Surgical and other treatment that is imperative is done. In the main, however, they rely upon the Dowling pack.

Dr. Myers demonstrated by means of the motion picture the meticulous care which must be followed in applying these packs if they are to be efficacious. These may be used in very young children. Any rhinologist may probably get a very excellent idea of the use of the Dowling pack by a careful study of the film shown by Dr. Myers, but it would seem difficult for even an experienced rhinologist to properly apply the Dowling pack without instructions from an experienced man or a thorough study of the film.

For acute conditions they use adrenalin; they do not use opiates under any circumstances except for operative procedures. They also emphasize the importance of stimulating elimination by all possible methods. The hot general pack, large quantities of water and regular bowel evacuation are emphasized.

They claim that by these methods sensitization phenomena are cured as well as are the inflammatory processes. In the individual whose asthma has not been of long standing they hope for a permanent cure. All cases are benefited. Even hay fever is usually cured, at least temporarily, by the procedures which they use.

They lay special stress upon the urine findings in regard to (1) acidity, (2) the indican, and (3) reducing power, which they reduce to a minimum as quickly as possible.

ST. JOSEPH'S HOSPITAL (Phoenix)

(February Staff Meeting).

The regular monthly meeting of St. Joseph's Hospital was held on Feb. 11, 1929, at eight o'clock with 29 members present. Dr. E. Payne Palmer, chairman, presiding.

Dr. Franklin H. Martin, president of the American College of Surgeons, Dr. Richard Smith of Grand Rapids, Mich., and Dr. Bowman C. Crowell, assistant director of the American College of Surgeons, were present as visitors.

The minutes of the last meeting were read by the secretary. The Hospital Analysis for the month of January was read and approved. The chairman called on each of the visitors to speak a few words.

Dr. Martin explained about the health inventorium which has been fostered by the College of Surgeons in connection with the Gorgas Memorial. Dr. Smith gave a resume of research work done by him on ectopic pregnancies during the last five years. Dr. Crowell complimented the staff on the reports of their meetings which have attracted attention, and also the contribution of the hospital to the committee on bone sarcoma in case reports submitted.

DR. FRED HOLMES had arranged a program on chest cases, and the following cases were discussed: reports submitted.

"Phrenicotomy." Cases 15417 and 15519. The following case history was presented by DR. VICTOR RANDOLPH as typical of those on whom phrenicotomy has been done.

Male, single, age 27. Chief complaint is cough and expectoration of about one ounce in twenty-four hours and attacks of fever coming on every few weeks to two or three months intermittently during the past two or three years. He has not been well for eight years. In 1921 he was diagnosed to have tuberculosis and since then has been pronounced practically arrested several times but each time has broken down after a few months of relatively good health. During the past two years he has been working at light work part time and has had repeated flare ups during which the temperature reached 103 or 104 for several days before subsiding. Pleurisy often accompanied the attacks. The expectoration has remained at about one ounce in twenty-four hours in the past year.

Physical examination shows well nourished man, not apparently in ill health. Temperature is 99.2, pulse 80. Examination of the head and neck, negative. The chest shows marked dulness over the entire left chest both front and back. There are numerous rales throughout the left. There are a few fine rales at the right upper front. Cardiac dulness shows heart apparently displaced to the left side. Examination otherwise negative. X-ray report is as follows:

"Roentgenogram of this chest shows on the right side fairly extensive infiltration density through the upper lung field as low as the fourth rib.

The left side shows almost total density with marked retraction of the heart, mediastinum and trachea. The lung detail is entirely hidden by fibrous pleura and heart shadow."

Fluoroscopic examination shows right diaphragm movable and the left diaphragm moves down about an inch and a half on deep inspiration, shadows of the heart overlapping diaphragmatic shadow.

In view of the marked density in the left with displacement of the heart, pneumothorax was not attempted and it was felt that phrenicotomy might offer some benefit. A phrenicotomy was performed on Jan. 11th, 1929; the left nerve was removed in its entirety through transverse incision under local. Skin clips were removed on the fifth day, incision healed. Patient was seen three weeks after opera-

A new milk modifier and diet supplement *with the added value of Vitamin B*

"I believe that every infant should have an addition of Vitamin B to its formula and should not depend on milk, either human or cow's , as its only source of this vitamin. Just as regularly as orange juice and cod-liver oil are prescribed, one should prescribe a substance rich in Vitamin B for the infant dietary."

J. Raymond Hoobler, M. D., Detroit
Journal A.M.A. August 4, 1928

The dangers from Vitamin B deficiency in artificially fed infants are apparent. Vitamin B is essential as a growth and antineuritic factor, as well as for adequate nutrition throughout life. The Vitamin B content of cow's milk is not constant and, when the milk is diluted, the vitamin content is necessarily reduced.

Even mother's milk may be very low in Vitamin B, particularly if the mother does not receive an adequate and balanced diet.

Quoting further from Dr. Hoobler's article:

"It is the consensus of opinion among the laboratory investigators in the fields of nutrition that Vitamin B is the dietary factor which most rapidly stimulates growth, and indeed that it may be the most important factor.

"Not only is this increase in growth brought about by stimulating the appetite and thus increasing the food intake, but even when the appetite is entirely satisfied and the individual remains on the same basal diet, an increase in Vitamin B substance will cause a gain in weight without necessarily increasing the intake of food. This would indicate that Vitamin B, in addition to stimulating the appetite, also brings about a better assimilation and utilization of the food intake."

To meet the need of pediatricians and general practitioners for a milk modifier dietetically suitable for the carbohydrate requirements of the infant, *and also possessin the added value of Vitamin B*, E. R. Squibb & Sons have developed Vitavose.

What Vitavose is

Squibb's Vitavose is a palatable and highly nutritious maltose-dextrin preparation, *exceedingly rich in Vitamin B* and assimilable iron salts. It contains not only maltose and dextrins but in addition all of the water-soluble extractives from the wheat embryo—Vitamin B, soluble nitrogenous compounds and mineral salts.

Wheat germs, freed from oil, are cooked, malted and extracted under conditions which preserve the important dietary properties and guarantee a maximum extraction of Vitamin B. The finished product, Vitavose, resembles a fine, golden yellow sugar with an agreeable malty taste.

What Vitavose is for

1. To modify cow's milk in infant feeding.
2. To supplement the diet of children, expectant and nursing mothers, invalids, convalescents and malnourished adults.
3. To supplement certain therapeutic diets where an abundance of Vitamin B and iron salts is indicated.

Because of its high Vitamin B content, Vitavose stimulates the appetite. Its slightly laxative qualities aid in overcoming a tendency to constipation.

Physiologically tested. Accepted by the Council on Pharmacy and Chemistry, American Medical Association

E. R. SQUIBB & SONS
Professional Service Dept.
80 Beekman St., New York

Please send me sample of Vitavose and detailed information.

Name .....

Address .....

SQUIBB'S VITAVOSE

Nearest Squibbs Biological Depot **728 South Hill St., Los Angeles, California**

tion, at which time left diaphragm is immobile and apparently elevated one inch or more.

Dr. Holmes discussed the indications for and technique of phrenicotomy or phrenic exeresis (as it is preferably called). He exhibited ten phrenic nerves which had been removed, and showed the variations in the origin of the accessory nerve. Two patients on whom the operation had been performed were shown and examined by the various doctors.

"Empyema with Multiple Abscess" was discussed by DR. S. I. BLOOMHARDT. He stated that the following history had been given him by Dr. R. W. Craig, who was in charge of this patient:

White male, age 35. Six weeks ago was exposed to fire and smoke. Shortly after this he was taken down with severe attack of influenza followed by pneumonia. He was attended by another phyisican and seen by Dr. Craig seven days after onset of illness. At this time the patient had a definite pneumonia in the right lower lobe. During the next 24 hours he developed an acute nephritis, urine showing albumin, hyaline, granular and blood casts and blood cells; there was almost complete suppression for a time. In about a week his right pleural cavity filled with fluid, proved by needle puncture to be pus. A rib was resected under local anesthesia, and a large amount of pus drained off. Several days after rib resection, two or three lumps appeared on each thigh, red and hot; these were incised and two or three ounces of pus were drawn off from each one. Blood culture was negative. At end of another week, a high temperature and thrombophlebitis of left arm above elbow developed. All pus was cultured and no other organisms than pneumococcus found; not typed. At the beginning of last week, temperature went to 103 and 104 again, with great deal of pain in both sides of chest; lower third of chest was nearly flat. Question in Dr. Craig's mind was whether to put in needle and aspirate, which he decided not to do. Several mornings ago, after severe coughing, dressings and bed were saturated with pus from empyema wound and sinus, followed by steady recovery.

Discussion of Empyema by DR BLOOMHARDT:

Allen made an important and interesting study in regards to the production of empyema and that is upon the importance of hemothorax in idiopathic and post-operative empyema. In his experimental operations upon the heart, Allen found that empyema developed in all of the animals in which there was marked soiling of the pleural cavity with blood. Accordingly, he began a series of experiments with this factor. If a simple suspension of hemolytic streptococcus was injected into the pleural cavity, there was a twenty-seven per cent incidence of empyema, but if a few c.c. of blood was introduced with the suspension, empyema followed in every case. In another series, a suspension of unattenuated pneumococci, type I, was injected in eight rabbits and only one developed empyema and in this animal it seemed very evident that there had been bleeding into the pleural cavity from the needle wound. In ten rabbits, blood also was introduced and everyone developed empyema and died. From these experiments, he justifies the theory that hemothorax plays an important part in the etiology of idiopathic empyema. In the streptococci group, he calls attention to the observation of McCallum that bloody fluid was aspirated almost universally during the first days of the development of infection in the pleural cavity. He found the same true in two cases of empyema due to the hemolytic strep, recently treated at the Barnes Hospital.

Patients dying from pneumococcal pneumonia not infrequently have granulation tissue on the visceral pleura. This furnishes a source of blood in this group.

In a report by Williams on the empyema group in adults at the Melbourne Hospital from 1919 to 1924, there were 139 cases, with 22 deaths,—16 per cent mortality. Williams, in treatment, does a rib resection and puts in a short drainage tube. He removes this in four or five days and for seven days after this, he spreads the wound with Spencer Wells forceps while the patient coughs. He claims if there should be a reaccumulation, it is always smaller and as he puts it, "A fresh start can be made with the smaller cavity along the same lines as before, with the addition of, perhaps, scraping the cavity gently with a Volkmann's spoon so that the lung is not hampered by organizing fibrins. He found if the tube was removed in seven days or less the average stay in the hospital was 5.4 weeks; if over eight days 7.4 weeks. He prefers local anesthesia.

Oelschlaegel reports 70 cases with 14 deaths,—20 per cent. Dr. Muller in his review of this paper noticed an interesting point when he—Dr. Oelschlaegel—says that if a given technic insures thorough drainage of the pus in empyema, the mortality associated with the use of that technic is of relatively little value as a criterion of the value of the method, as empyema is often only a complication of some basic disease having no therapeutic relation to it.

Heiles—another Australian—writing on this subject, states that he practically never saw bronchial fistula in these empyemas and never had a case of chronic tuberculous empyema.

Last yeaar, Bohrer of the Bellevue Hospital, New York City, gave a report of the study of 154 cases of empyema in children. Also, Downes of Melbourne, Australia, a series of 446 cases.

Lobar pneumonia is the primary factor in the great majority of these cases. In so many cases, the empyema can only be regarded as one factor and often a minor factor in the clinical picture. Double pneumonia was present in four per cent of the series and the mortality was 72 per cent. Bohrer follows the usual plan of not operating during the formative stage unless there is distress, in which case repeated aspirations may be necessary. When the fluid turns to pus and the pneumonia has subsided he institutes intercostal drainage. Local anaesthesia is used and a small incision made in the seventh intercostal space near the midaxillary line, but the site may vary with the location of the pus. In a few days, irrigation is started with saline solution, and if no coughing is produced, Dakin's is then used cautiously. If after the end of 8 or 10 days the child has not improved and there is no acute otitis media or recurrence of the pneumonic process, he concludes he is dealing with a complicated form of suppurative pleurisy. Then an exploratory thoracotomy under light ether or gas—an intercostal incision is made long enough to give proper exposure and a rib spreader is introduced. If more exposure is required, a rib may be cut at one or both ends and telescoped. This gives the advantage of visual guidance, in dividing vicious adhesions, doing a partial decortication or any other procedure necessary, thus converting multiple loculi into a single cavity, or placing drainage when it is needed. Bohrer states this may appear to be radical surgery but, properly done, it produces little shock, reduces mortality, shortens convalescence and causes no deformity. He states also that the intercostal incision alone will cure about 80 per cent of the cases. He considers skillful nursing, high caloric diet and blood transfusion as highly important in the after treatment. He always gives blood transfusion in the case of the second operation.

"Lung Abscess," with presentation of patient. DR. KIMBALL BANNISTER.

NOW READY

We have just issued

A NEW PAMPHLET

devoted to a discussion of May, June, and July

HAY FEVER

and in this bulletin the chief causative plants are illustrated in natural colors.

Copy sent on request

THE ARLINGTON CHEMICAL COMPANY

YONKERS, N. Y.

Case No. 15521, "Hemothorax of Uncertain Etiology," presented by DR. HOWELL RANDOLPH, as follows:

Unmarried female, age 23, came to us complaining the left side of the chest.

P. H. Had always been very healthy. Had the "flu" in 1918, severe attack. Measles at 18 years, severe.

Present Illness. She had not been well for the past two years. Began with what she called a nervous breakdown, but at that time she had a small hemorrhage from the lungs, presumably, and following this she was in bed for two months, then got up and went back to school. She seemed strong, and taught school winter of '27-28. Had a slight hemorrhage in Nov. '27. Another hemorrhage occurred in Feb. 1928 and small ones in July and again in November 1928.

She has remained in bed since Aug. 1928, and thought she was doing well, but two weeks ago she had a reverse, with high temperature up to 103, and marked shortness of breath. At the same time she believed that the expectoration was somewhat reduced. Sharp pain was very annoying, present to the left of the sternum, severe two weeks ago and still soreness is present. She has become very much weaker since this attack two weeks ago. Temperature has continued to run high.

Physical Examination. Temp. 101., appears "toxic"; lies on her back with least discomfort. Quite pale. The skin has a yellowish tint. Heart dullness extends well over to the right of the sternum; sounds are clear. Lungs show dullness to flatness from apex to base of the left lung, anteriorly and posteriorly. Breath sounds diminished to absent over the left base, and markedly increased in volume over the upper. Amphoric quality, above the fourth rib. Exaggerated breath sounds on the right side. Area of triangular dullness to the right of the midline posteriorly, at the base. Fingernails curved.

X-ray examination shows density over the entire left basal area, with displacement of the heart to the right. There certainly seems to be no other good explanation for this finding except fluid. The density shades off gradually into the apex.

Aspiration was attempted on four different occasions, at different levels, using various sized needles up to a trocar of about 3 m.m. bore. The specimen obtained each time was dark red blood, which was very difficult to draw through the needle, and not more than three or four c.c. was removed each time, because the blood had evidently clotted within the pleural cavity. Pneumococci were present in each specimen on smear, but no growth on culture except one time, when staphylococci were found, possibly contamination.

Repeated sputum examinations have shown no tubercle bacilli, although the expectoration remains at one to two ounces daily, and concentration method was used on one specimen.

The blood count shows Hb. 60, white count 7,000 to 12,000, red count 3,200,000; polynuclears 71 per cent, mononuclears 29. Red cells show moderate achromia.

Coagulation time on two occasions was 9 min. and 10 minutes by the capillary method.

Blood cultures were sterile. Blood calcium was 9.8 mgs. per 100 c.c. Blood type Group II.

Summary:. In view of the history of repeated hemorrhages with attacks of fever over a long period of time, of having an x-ray in Feb. 1928, which was said to have shown tuberculosis, it would seem that the probable primary pathology was a tuberculosis of the lungs. No data at present at hand gives us this diagnosis definitely however. The sputum has been repeatedly negative for tubercle bacilli, the roetgenographic examination shows the right side to be clear, with the primary pathology on the left being obscured by fluid.

Assuming then, that the patient has tuberculosis, we feel that the probable diagnosis in this case is pulmonary tuberculosis, and spontaneous pneumothorax occurring about three weeks ago, with hemorrhage in the pneumothorax cavity.

Treatment: So far the treatment has consisted of repeated attempted aspiration, and general supportive therapy, in the way of diet, rest and relief of discomfort.

DR. H. P. MILLS, pathologist, reported upon nine autopsies performed during the month of January, as follows:

Case 15439:- Male, age 54, married, entered Dec. 24, 1928, with diagnosis of tuberculosis of epididymes and seminal vesicles. Operation of vasotomy and bilateral epididymectomy. Death on Jan. 15, 1929.

Autopsy:- Lungs normal without gross signs of tuberculosis. Heart normal. Liver enlarged to about one and a half times normal size. Gall bladder thickened and filled with thick, dark green bile. Intestines and stomach covered with plastic exudate, mesentery pushed to one side, lying on right side below liver. Large amount of free exudate in peritoneal cavity. Appendix retrocecal, fibrous and adherent.

Right kidney immense, polycystic, filled with purulent material; right ureter widely dilated, walls thickened, kinked in several places and filled with same purulent material. Left kidney practically same as right, though left ureter not dilated. Bladder filled with purulent material.

Diagnosis:- Renal tuberculosis; peritoneal tuberculosis.

Case 15363:- Male Mexican, age 45, entered Jan. 5, 1929, with diagnosis of poisoning by some unknown substance. Died on Jan. 7th in epileptiform attack following maniacal periods. Spinal fluid was negative, and blood findings negative; white count 16,700.

Autopsy:- Lungs and heart normal. Kidneys, spleen and intestines were normal. Head not examined. Stomach normal. Chemical analysis of stomach or tissues not made.

Cause of death not shown by autopsy.

Case 15378:- Two-year-old female child entered Jan. 8, 1929, with laryngeal diphtheria. In spite of antitoxin and supportive measures, child died the next day.

Autopsy was done, removing the entire contents of the thorax for detailed examination. Larynx and trachea lined by membranous exudate extending as far as the bifurcation, evidently a diphtheritic membrane. Sections of the kidney showed degenerative changes in the tubular epithelium to an extensive degree, evidently a toxic nephritis.

Cause of Death: Diphtheria (laryngeal) with toxic nephritis.

Case 15320:- Male, age 68. History of pain coming on two years ago extending from third rib upward over shoulder, dull and intermittent. Two months later began to lose weight and strength, with gastro-intestinal upsets in form of nausea, vomiting, small gray colored stools. Special diet did not improve symptoms, and pain continued with symptoms of chronic bronchitis. Entered the hospital on Dec. 25, 1928, markedly emaciated, with extreme loss of strength. Cough was distressing and on one occasion more than a drachm of blood was expectorated. Physical findings were negative except for marked dullness at left apex down to sixth interspace. Diminished breath sounds and moist rales at base of right lung.

X-ray examination showed no outline of gall-blad-

A New Idea of Special Interest to Obstetricians

The CAMP

MATERNITY BRASSIERE

and Breast Support

To render a more complete service in our Maternity Garments, we offer to the profession a very efficient Maternity Breast Support for use both before and after parturition.

In design this garment carries into effect the Camp System of Adjustment, which gives a simple way of adapting size to changing body proportions. It prevents sagging of muscles, and acts as a "sling" for the breast. It also assists in restricting the accumulation of superfluous fats throughout the upper body.

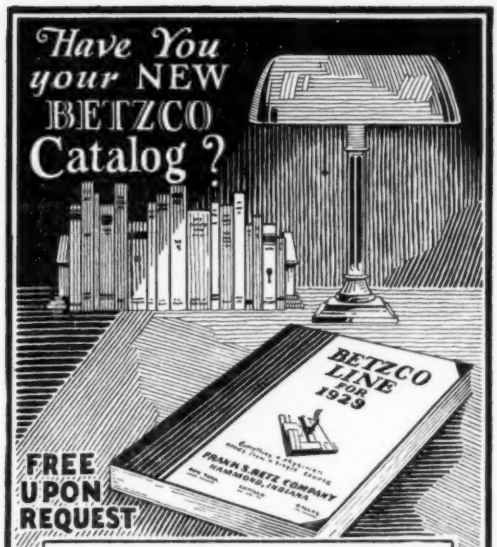
In purpose, this garment correlates perfectly with our Camp Maternity Abdomnal Supports.

S. H. Camp and Company

Manufacturers—Jackson, Michigan

New York City 330 Fifth Ave. Chicago 59 E. Madison St. London 52 Mortimer St.

Have You your NEW BETZCO Catalog ?



FREE UPON REQUEST

FRANK S. BETZ COMPANY NEW YORK CHICAGO DALLAS
HAMMOND, INDIANA

Send me my copy of the "BETZCO LINE" for 1929

Name .....

Address .....

City ..... *State* .....

As a General Antiseptic

in place of

TINCTURE OF IODINE

Try

Mercurochrome-220 Soluble

Dibrom-oxymercuri-fluorescein

2% Solution

It stains, it penetrates, and it furnishes a deposit of the germicidal agent in the desired field.

It does not burn, irritate or injure tissue in any way.

HYNSON, WESTCOTT & DUNNING

Baltimore, Maryland

"UNIVERSAL" GLOLITE

Perfected

Infra-Red energy is a great deal more convenient than any other form of heat.

Infra Red LAMPS

More Even Distribution

Deeper Penetration

It Makes a Difference Who Builds the Infra-Reds You Buy!

Be sure to compare Glolites before you buy. They are made in several styles ranging in price from $5 to $27.50

Glolites are of the very finest quality and were the first ROUND Infra-Red Generators offered to the Profession. Practically all manufacturers have adopted SOME TYPE of round generator

Many consider "Glolite" Infra-Red radiation actually better than diathermy for treatment within three inches of the surface.

The Lamp is efficient—beautiful in design—Sturdy in construction and makes a most impressive appearance in any doctor's office.

MANUFACTURED BY

PAUL E. JOHNSON, Inc.

MERCURY QUARTZ LAMPS CARBON ARC LAMPS
RADIANT THERAPY LAMPS INFRA RED LAMPS

1854-70 SOUTH ALBERT STREET

Chicago, U. S. A.

Write us Or Any Authorized Dealer

der by Graham dye test; barium meal examination of stomach was negative. Roentgenogram of lungs showed "total density over the upper left lobe, density being fairly well defined, but not resembling lobar consolidation. Heart is enlarged and there is well marked irregular density through the lower left lung field. Shadow in the upper left would be either new growth or an irregular type of inflammatory consolidation, with preference for diagnosis of new growth."

Death on Jan. 3, 1929.

Autopsy: White male, apparently 65 years of age, weighing approximately 140 pounds, in moderate state of emaciation.

Lungs: Left pleural cavity had approximately 100 c.c. of seropurulent basal effusion. Apex adherent, solid, without apparent nodules. Cross section showed tumor growth extending well into the parenchymal structure of upper three-fourths of upper lobe, apparently arising from the upper main bronchus. Base showed moderate degree of red hepatization, as did the right lung. Pericardium showed 10 to 15 c.c. of straw-colored fluid. Myocardium not definitely changed Moderate sclerosis of coronary vessels. Moderate sclerotic changes in the valves; slight dilatation of aortic ring with atheromatous change about the sinus of Valsalva and aortic arch.

Stomach normal. Liver shows moderate cirrhosis. Spleen normal. Small and large bowel negative. Appendix small, fibrotic and adherent in retrocecal fossa. Gall bladder showed marked inflammatory changes with thickened wall and two large stones with several smaller stones. Pancreas fixed with fibrotic changes about head; tail fibrosed with calcium deposit. Kidneys small and contracted; double ureter on the left side. Bladder negative. Prostate showed considerable hypertrophy but no nodules and no evidence of malignancy.

Anatomical Diagnosis: Primary carcinoma of left upper lobe of lung (histologic section). Pneumonia in stage of red hepatization. Cirrhosis of liver; chronic interstitial nephritis; cholelithiasis; chronic pancreatitis; chronic appendicitis.

Case 15308: Married woman, age 52. Entered Dec. 29, 1928 with diagnosis of broncho-pneumonia. No history provided by attending physician, and no record of physical findings. Death on Jan. 3, 1929.

Autopsy: Cellular layers of skin showed blue color, htis color appearing eight hours after embalming. (Note: Probably due to methylene blue given prior to entrance to hospital.)

Left lung; hyperemia of lower lobe; multiple dense pinhead white nodules mostly in pleural surface; empysema of upper left lobe. Right lung covered with recent fibrous exudate and adherent pleura. Weighs four times as much as left lung. Lobar consolidation of right upper and lower lobes in stage of gray hepatization; middle lobe not involved. Pericardium and aorta blue. Heart normal except for narrowing of mitral valve due to fibrous thickening. Left ventricle hypertrophied. Lower aorta shows atheroma. Liver and spleen and intestines normal. Liver enlarged and surface mottled. Gall bladder enlarged and thickened. Left kidney very large, cortex narrowed, capsule adherent. Right kidney atrophied with stricture of right ureter.

Anatomical Diagnosis: Lobar pneumonia of right lung; chronic cholecystitis; hypertrophic cirrhosis of liver; arteriosclerosis.

Case 12068: Married woman, age 37, entered on Jan. 11th, for third time, with diagnosis of bronchial asthma. X-ray examination of sinuses showed dense antra.

Physical examinations are recorded and indicate failing heart. Patient died on Jan. 13th.

"Particular Prescriptionists"

Distributors for

Parke, Davis & Co's.

BIOLOGICALS and AMPOULES

Sufficient Stock for All Emergencies

Fresh Stock of

Rabies Treatment

Cumming Method

Manufactured by

Parke, Davis & Co.

On Hand at All Times

Mail, Telephone and Telegraph Orders Given Immediate Attention

Wayland's Central Pharmacy

Goodrich Block

PHOENIX, ARIZONA

Autopsy: Lungs showed marked emphysema with slight hyperemia of lower lobes and patchy areas of active hyperemia more dense than surrounding tissue, apparently beginning consolidations, probably influenzal type of broncho-pneumonia. Heart slightly enlarged; all the cavities filled with postmortem clot, extending beyond the arch of the aorta. Mitral valves show fibrous thickening, other valves normal. Marked passive hyperemia of liver with characteristic nutmeg appearance; membrane pals and friable. Marked passive hypermia of kidneys, and veins of abdomen all distended.

Section of lung tissue showed marked thickening of walls of alveoli, resembling a chronic emphysema. Extensive infiltration with red blood cells and exfoliated endothelium in the air sacs, forming a partial consolidation. Section of kidneys showed marked hyperemia of glomeruli and kidney parenchyma. Section of liver showed chronic passive hyperemia.

Anatomic Diagnosis: Broncho-pneumonia; heart failure.

Case 15591: Male, age 46, married. Entered Jan. 27th. History of illness beginning on Thanksgiving Day, with chill, fever, loss of appetite and vomiting; diagnosed pneumonia of left lung. Partly recovered and went to Santa Fe, N.M., ten days ago, where he developed pneumonia again, on the right side this time. Brought here on train from Santa Fe.

Examination showed an extremely ill man, sitting propped up in bed, responding sluggishly to questions. Edema of right side of face. Rales and bronchial breathing over entire right side and mucous rales in left base. Heart rate irregular. Right arm swollen; feet and ankles edematous. Urine showed positive albumen, hyaline and granular casts; white count 15,200. NBN 75 mgm., urea nitrogen 38 mgm.

X-ray examination showed heart enlarged to left and irregular density through lungs interpreted as unresolved pneumonia.

Patient died on Jan. 30th.

Autopsy:

Lungs: Dense fibrous adhesions near the apex on each side, and left lung was adherent to the pericardium. Left lung showed marked irregularity in contour and was very dense. The cut surface showed marked fibrous-like induration throughout lower part of lung and in two or three areas in lower lobe there was recent consolidation, showing stage of red hepatization. The right lung showed consolidation of the middle lobe in stage of red hepatization. Upper and lower lobes showed marked irregular distribution of fibrous induration.

Heart was markedly enlarged, tricuspid valve widely dilated and incompetent. The right ventricle walls were thinned; the mitral valve showed fibrous thickening but no induration; other valves normal. There was moderate amount of atheroma of the lower portion of the aorta.

Gastro-intestinal tract was negative.

Spleen markedly enlarged, quite friable and showed marked hyperemia. Both kidneys enlarged, and on gross section showed marked passive hyperemia. Liver was somewhat reduced in size, showed mottled appearance both on the surface and on cross section, having the characteristic appearance of "nutmeg" liver of chronic passive hyperemia.

Section of liver showed extensive hemorrhage infiltration with atrophy of parenchymatous cells, changes characteristic of chronic passive hyperemia.

Founded 1896 by Dr. Hubert Work

WOODCROFT HOSPITAL, PUEBLO, COLORADO

New Building
New Equipment

NEURO-PSYCHIATRIC
CLINIC
NERVOUS AND MENTAL
DISEASES
DRUG ADDITIONS

H. A. LaMoure, M. D.
Superintendent

Calcreose

—a Good Expectorant
—a Stimulant for the Appetite
—a Form of Creosote that Patients Will Tolerate
—a Valuable Adjunct in Treating Tuberculosis
—an Effective Remedy in Bronchitis, Stubborn Coughs and Respiratory Ailments

Calcreose can be given in large doses for long periods without apparent difficulty

Samples of Tablets to Physicians on Request

THE MALTBIE CHEMICAL CO. Newark, New Jersey

Manufacturers of Pharmaceutical Products

Section of spleen shows very marked hyperemia with hemorrhagic extravasation.

Section of kidney show injection of the glomerular vessels and cloudy swelling and desquamation of the tubular epithelium,—chronic passive nephritis with excessive hyperemia.

Section of lung show very much thickening of pleura and excess of fibrous structure throughout. The air vesicles in this section are partly filled with desquamation of epithelium and red blood cells—chronic fibrosis of lungs with recent pneumatic consolidation.

Diagnosis: Incompetence of tricuspid valve and dilated right ventricle; chronic induration of both lungs, with acute bronchial pneumonia; passive hyperemia of spleen, kidneys and liver.

Case 15581-A:- Baby, 19 mos. old, entered Jan. 28th, with diagnosis of broncho-pneumonia, dying before examination could be made.

Autopsy:-

Lungs: Hyperemia both lower lobes and posterior portion upper lobe on right. Distributed through these lobes were irregular patches of much deeper and darker discoloration, having the appearance of hemorrhagic infiltration and where these extended to the surface there was definite pitting of the pleural surface, evidently due to failure of air to reach this portion of the peripheral lung. These changes were considered to represent an acute broncho-pneumonic process probably influenzal in type.

Heart: Normal in gross appearance.
Gastro-intestinal tract negative.
Kidneys, spleen, normal.
Liver: No definite gross changes were found.

Gall bladder somewhat thickened—otherwise negative.

Head was opened: Brain removed. No excess amount of fluid in the arachnoid space. There was rather marked hyperemia but no gross hemorrhage or evidence of meningitis.

Cause of death: Early broncho-pneumonia, probably associated with influenza.

Case 15550. Female, married, age 55. Entered hospital Nov. 24, 1928, with history of having suffered from indigestion and nausea for several years. Chief complaint at this time was vomiting of blood. Mass could be felt in upper left abdomen. Brought to hospital for diagnosis.

X-ray examination showed extensive involvement of fundus and pylorus, with characteristic appearances of carcinoma, with advanced degree of obstruction. Some x-ray therapy was given in hope of securing some relaxation of pylorus, but this did little good. Patient gradually grew worse and died on Jan. 24, 1929.

Autopsy: Entire stomach was involved, hard and cartilaginous, with extension into lower esophagus. Omentum was involved with consistency of pasteboard.

Final Diagnosis: Advanced carcinoma of stomach with many metastases through abdomen.

The following testimonial relative to Dr. Charles S. Vivian, deceased, was adopted by vote of the staff:

The Medical and Surgical Staff of the Hospital wish to record their deep apprecation of the professional attainments of Dr. Charles S. Vivian, and the great loss sustained by the staff and hospital, in his death. Through his efforts and because of his work, the specialty in urology was brought to a high degree of efficiency in this hospital, as well as in his private work. In his daeth, the staff has lost a valuable member, and a real contributor to medical and surgical progress. The members of the staff extend to the bereaved wife and children sincerest sympathy and hereby record our deep appreciation of the loss sustained by family,, hospital and community.

D. FOURNIER, Sec'y.

BOOK REVIEWS

THE TECHNIC OF LOCAL ANESTHESIA.—Arthur E. Hertzler; A.M., PH.D., LH.D, F.A.C.S.; Professor of Surgery in the University of Kansas; surgeon to the Halstead Hospital, Kansas; to St. Luke's Hospital and St. Mary's Hospital, Kansas City, Missouri; and to the Providence Hospital, Kansas City, Kansas. Fourth Edition. 146 illustrations; St. Louis, The C. V. Mosby Company; 1928; $6.00.

This is the fourth edition of this excellent work. The fact that there have been four editions in the last several years seems ample proof of its popularity. The author has handled his subject with his usual thoroughness. He attempts to solve, not what operations can be done under local anesthesia, but what operations can best be done. The indications for the use of the local anesthesia together with the study of their contraindications plus the skill of the operator are the two dominant factors in the use of local anesthesia. The few number of deaths under local anesthesia is discussed at length. No conclusions as to why the

SAVE MONEY ON
YOUR X-RAY SUPPLIES

Get Our Price List and Discounts Before You Purchase

WE MAY SAVE YOU FROM 10% TO 25% ON X-RAY LABORATORY COST

Among the Many Articles Sold Are

X-RAY FILM—Buck X-Ograph, Eastman or Agfa Superspeed Duplitized Film. Heavy discounts on standard package lots. Buck X-Ograph, Eastman and Justrite Dental Films. Fast or slow emulsions.

BRADY'S POTTER BUCKY DIAPHRAGM insures finest radiographs on heavy parts, such as kidney, spine, gall-bladder or heads. $250
Curved Top Style—up to 17x17 size cassettes.....$250.00
Flat Top Style—11x14 size, $175.00; 14x17 size $260.00

BARIUM SULPHATE. For stomach work. Finest grade. Low price. Special price on 100-pound lots.

DEVELOPING TANKS, 4, 5 or 6 compartment stone, will end your dark room troubles. Ship from Chicago, Brooklyn, Boston or Virginia. Many sizes of enameled steel tanks.

DENTAL FILM MOUNTS. Black or gray cardboard with celluloid window or all cellulod type, one to fourteen film openings. Special list and samples on request. Either stock styles or imprinted with name, address, etc.

INTENSIFYING SCREENS—Buck X-Ograph, Patterson or E. K. Screens, for fast exposure, sold alone or mounted in cassettes. Liberal discounts. All-metal cassettes in several makes.

If you have a machine have us put your name on our mailing list.

GEO. W. BRADY & CO.
790 S. Western Ave., CHICAGO

deaths should have occurred is possible to be drawn. The various agents used for local anesthesia are discussed in detail. Novocaine is the almost universal choice and has a wide field of applicability. It is only one seventh as toxic as cocaine. Quinine has a limited but useful field. He discusses technic of administration, general operations, operations o nthe scalp, cranium and brain, on the ear and mastoid, gasserian gangloin, tonsils, adenoids, larynx, trachea, thyroid gland, mammary gland, thorax, lungs and spine, abdominal operations, hernia, bladder, urethra and prostate, female organs, rectum and extremities.

This book should appeal to every surgeon.

INJECTION TREATMENT OF INTERNAL HEMORRHOIDS.—Marion C. Pruitt, M.D., L.R. C. P., S. (ed.) F. R. C. S. (Ed.) associate in Surgery, Medical Department, Emory University, Georgia Baptist Hospital and Grady Hospital; proctologist, Davis-Fischer Sanitarium and Anti-Tuberculosis Association; formerly Resident Surgeon, Westminster Hospital, London, England; Lieutenant, Temporary and Honorary Commission, R.A.M.C.; Major U.S.M.C. Illustrated. St. Louis. The C. V. Mosby Company; 1829; $3.00.

The author has written a small book giving a terse valuable discussion of the anatomy, physiology and pathology of the anus as regards hemorrhoids.

He discusses in detail the history of the injection treatment of hemorrhoids. He gives the various agents and methods which have been used.

During the World War it fell to the author's lot to treat a large number of cases of hemorrhoids. He used the injection treatment and was gratified with the results. He has continued the treatment since the war and has used it in a large number of cases and is enthusiastic about the results.

In the last chapter he gives a series of cases which give the reader a conception of the results which may be expected.

BULLETIN OF THE NEW YORK ACADEMY OF MEDICINE: November 1928. Published monthly by The New York Academy of Medicine; Fifth avenue and 103rd Street, New York, N. Y.

A symposium is presented in this issue on "the problem of ageing and of old age." The following are the more interesting topics discussed, with the author of each given. The pains, penalties and prohibitions of old age. Can they be prevented? Sir Farquhar Buzzard. Old age and what it means to the community, Louis I. Dublin; Dietetics in old age, Samuel A. Brown; Pharmacology in old age, Alexander Lambert; Alcohol and old age, Samuel W. Lambert; The ageing of the human brain, Frederick Tilney; The mechanism of senescence, Alexis Carrel; and The ageing of the heart muscle regarded from a general biological point of view, Alfred E. Chon.

The perusal of these articles is worth the time of any physician.

WHEN ON THE ROAD TO RECOVERY

CONVALESCENCE demands the utmost in recuperative power . . . That is why Horlick's the Original Malted Milk is used with such universally good results when the patient is on the road to recovery.

It supplies nutrients most needed for the rebuilding of health and strength. By the exclusive Horlick process, these food elements are rendered easily and quickly assimilable. For samples, address—HORLICK—Racine, Wis.

THE ORIGINAL MALTED MILK

HORLICK'S

The G. Wilse Robinson Sanitarium and Neuro-Psychopathic Hospital

For Nervous and Mental Disorders and Allied Conditions

Alcoholism and Drug Addiction

Pleasantly located, on a beautiful tract of 25 acres. Buildings are commodious and attractive. Rooms with private bath are available.

Approved diagnostic and therapeutic methods used.

Occupational therapy, recreation and entertainment.

G. Wilse Robinson, M. D., Medical Director

Office:—Suite 814-817 Medical Arts Bldg. 34th and Broadway.

Sanitarium:—8100 Independence Road, Kansas City, Missouri

IMPERATIVE TRAUMATIC SURGERY—with special reference to AFTER CARE AND PROGNOSIS—by C. R. G. Forrester, M.D., F.A.C.S. Consultant, Teaching Staff, Illinois Post-Graduate School, Laboratory of Surgical Technique, Chicago; Consulting Staff, Chicago General, and Rogers Park Hospital; Attending Staff, West Side, Lutheran Memorial, and West Suburban Hospitals; Chicago. Illustrated. Paul B. Hoeber, Inc.; New York; $10.00.

If there are any other kinds of accidents which can happen to the body, than the author has included in his book, I cannot think of them, unless it is blood vessel injuries.

The book is profusely illustrated with excellent pictures, drawings, photographs and x-rays. There are 429 pages in the book; 21 chapters and 598 illustrations.

The book is a presentation of personal experiences and the author says he has used only those experiences in which the results were checked up.

He has paid special attention to immediate treatment and after-care of all traumatic conditions.

He has used the term "Imperative Traumatic Surgery' in preference to "Industrial Surgery."

The book seems to be complete. It is certainly clear and is not verbose. Every surgeon doing traumatic surgery ought to have this book in his library.

A Text-Book of Surgery. By W. Wayne Babcock, M.D., F.A.C.S., Professor of Surgery and of Clinical Surgery in the Temple University, Philadelphia; Surgeon to the Samaritan Hospital and to the American Hospital for Diseases of the Stomach. Octavo of 1367 pages; with 1050 illustrations, 9 of

The California Tuberculosis Association

Announces a Summer Course for the Study of Tuberculosis under the direction of

Dr. Allen K. Krause, Johns Hopkins Medical School

Los Angeles July 15th to 26th

San Francisco July 29th to August 9th

Enrollment limited to 25 in each place. Application for enrollment should be made to the California Tuberculosis Association, 901 Griffith-McKenzie Building, Fresno, California. Fee $25.00 Program will be published at a later date.

Nonspi

(An Antiseptic Liquid)

For Excessive Armpit Perspiration

Keeps the underarms dry and odorless.

Samples mailed on receipt of this coupon.

THE NONSPI COMPANY
2652 WALNUT STREET
KANSAS CITY, MISSOURI

Send free NONSPI samples to:

Name.....

Street.....

City.....

NONSPI

From Text Books of Over a Decade

MEAD'S DEXTRI-MALTOSE

FOR more than twenty years dextrin-maltose has been cited in text books of leading authors on infant feeding. During this period, no reversal of opinion has occured, and the opinions set out by the earlier writers are shared by those of today.

This form carbohydrate in the combination of Mead's Dextri-Maltose is usually the sugar of first consideration where the infant's diet is one of diluted cow's milk with carbohydrate additions.

From Text Books of over a decade

For years it has been indicated by physicians both for the routine feeding of well babies, and in corrective diets for the treatment of nutritional disturbances.

MEAD JOHNSON & COMPANY
EVANSVILLE, INDIANA

them in colors. Philadelphia and London: W. B. Saunders Company, 1928. Cloth, $10.00 net.

The author has covered the entire field of surgery in the one volume of 1367 pages. The book is a shade over two inches thick and is easily handled. The publishers have used an excellent grade of paper. The numerous cuts are introduced directly in the text, so they add no excess bulk to the volume. The cuts are excellent.

The author has a terse understandable style, presenting much inlittle space. He presents his subject as a precise and exact science. In his preface, he says: "The student or physician who reasons 'Why learn today what tomorrow may prove wrong? Why strive for exactness when the most learned are often in error?" Not only loses his usefulness in a progressive profession, but lays down the torch of progress bequeathed to him. I have, therefore, written in a dogmatic vein what practice and study have made me believe is true today."

This is a book of such practical value that every general practitioner and every surgeon will wish to have it on his desk.

SUMMER CLINICS, CHICAGO MEDICAL SOCIETY

The Chicago Medical Society will hold a two-week's clinics at Cook County Hospital June 17 to 29 inclusive. Members of the hospital staff will give these clinics on the following schedule:

8 to 10 a.m.—Medical and surgical clinics in amphitheatres.
10 to 12 a.m.—Ward walks.
12 to 1 a.m.—Luncheon.
1 to 3 p.m.—Medical and Surgical clinics in amphitheatres.
3 to 5 p.m.—Ward walks.

The amphitheatre work will be devoted to medical and surgical dry clinics and lectures. Two amphitheatres will be used simultaneously, one for medical and one for surgical clinics. Each clinic will be one hour in length, thus giving four medical and four surgical clinics daily. Operative work will be done during the hours devoted to ward walks. The clinical work will be confined largely to general medicine and surgical subjects.

It is planned to hold six meetings to be addressed by speakers other than members of the hospital staff on such subjects as heart disease, tuberculosis, obstetrics, physiotherapy, gastro-intestinal disorders and possibly diabetes.

A reservation fee of ten dollars will be charged to cover the cost of preparing for and conducting the clinics.

For further information apply to the Chicago Medical Society, 185 N. Wabash Ave., Summer Clinics Committee.

WANTED

An association or placement with a group or an institution as director of physical therapy by an ethical M. D. of many years experience in this work. Full time service preferred.

Address A. M. C., care of Southwestern Medicine.

SITUATIONS WANTED

WANTED—Salaried appointments for Class A Physicians in all branches of the Medical Profession. Let us put you in touch with the best man for your opening. Our nation-wide connections enable us to give superior service. Aznoe's National Physicians' Exchange, 30 North Michigan, Chicago. Established 1896. Member The Chicago Association of Commerce.

ANNOUNCING

a change in name

HALEY'S M-O

is now known as

MAGNESIA-MINERAL OIL (25) HALEY

Accepted for N. N. R.

of the American Medical Association

FORMULA:

Each Tablespoonful Contains Magma Mag. (U. S. P.) 3 iii i, Petrolat. Liq. (U. S. P.) 3 i.

A uniform, permanent, unflavored emulsion of Liquid Petrolatum (U. S. P.) and Magma Mag. (U. S. P.). Does not disturb digestion, or irritate. For internal administration in indicated conditions and as an ANTACID MOUTH WASH. From several questionnaires sent out to thousands of physicians who have used this product, the following indications for its use in conjunction with other treatment have been compiled:

Oral or Gastro-intestinal Hyperacidity, Gastro-intestinal Fermentation, Gastric or Duodenal Ulcer, Intestinal Stasis, Obstipation, Autotoxemia, Hemorrhoids, Colitis, Ante- or Post-Operative, Pregnancy, Maternity, Infancy, Childhood, Old Age.

Generous sample and literature sent to any physician on request.

The
HALEY M-O COMPANY, *Inc*
Geneva, New York

PARKE, DAVIS & COMPANY

Why PARKE-DAVIS AMPOULES?

IT IS not practicable for the physician to test his ampoule solutions chemically or biologically before use; he must choose a manufacturer in whom he has faith.

In the manufacture of the Parke, Davis & Co. Ampoules the following problems have been met and mastered:

1—*Form.* Aqueous or saline solution, or suspension in a readily absorbable oil.

2—*Sterilization.* Not always a simple matter. Some chemical combinations are injured by heat.

3—*Standardization.* Both chemistry and pharmacology contribute.

4—*Stability.* A question of purity *and* chemical balance.

5—*Preservation.* In alkali-free glass—none other.

6—*Acid-base Equilibrium* (hydrogen-ion concentration). Assured by potentiometer tests.

Parke, Davis & Co. Ampoules for subcutaneous or intramuscular use are supplied in boxes of 6 or 12 and 100; for intravenous use in boxes of 6 and 25.

Ask for our Ampoule Booklet

PARKE, DAVIS & COMPANY
DETROIT, MICHIGAN